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Table of Contents

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ORIGINAL ARTICLES— PAGE.
An Address, by F. ST. JOHN POOLE, M.B., B.S. .. 395
"Simulation of Vascular Disease of the Geniculo-Calcarine Pathway by Cerebral Tumour", by KENNETH B. NOAD, M.B., M.R.C.P. 400
"Some Experimental Work on Poliomyelitis", by ROBERT SOUTHBY, M.D., B.S., and MARGOT MCKIE, B.Sc. 404
"The Use of Iodine in Thyreotoxicosis", by HUGH R. G. POATE, M.B., Ch.M., F.R.C.S., F.R.A.C.S. 411

REVIEWS—
Cardio-Vascular Pain and Biochemistry 414

NOTES ON BOOKS, CURRENT JOURNALS AND NEW APPLIANCES—
"Clinical Science Incorporating Heart" 414

LEADING ARTICLES—
Hospitals and the Depression 415

CURRENT COMMENT—
Scarlatinal Purpura 416
Tumours of the Suprarenal Gland 417

ABSTRACTS FROM CURRENT MEDICAL LITERATURE—
Morbid Anatomy 418
Morphology 419

BRITISH MEDICAL ASSOCIATION NEWS— PAGE.
Meeting of the Federal Council 420
Annual Meeting 423
Nominations and Elections 426

POST-GRADUATE WORK—
Annual Refresher Course in Melbourne 427
Post-Graduate Course in Obstetrics 427

THE ROYAL AUSTRALASIAN COLLEGE OF SURGEONS 428

UNIVERSITY INTELLIGENCE—
The University of Sydney 428

CORRESPONDENCE—
Cancer 428
Shall the Tonsil be Our Totem? 429
A Protest 429
The Treatment of Pneumonia 429

OBITUARY—
Albert Curtis 430
Lindsay Walker Craig 430

DIARY FOR THE MONTH 430

MEDICAL APPOINTMENTS VACANT, ETC. 430

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 430

EDITORIAL NOTICES 430

An Address.[1]

By F. ST. JOHN POOLE, M.B., B.S. (Adelaide),

Retiring President of the South Australian Branch of the British Medical Association.

I FOUND it a little difficult to choose a subject for the address which I am about to deliver and I am faced with still greater difficulty in dealing with my choice in a manner worthy of the audience of so important an assembly.

I might perhaps have found it easier to address you upon some scientific subject, but scientific subjects have been dealt with more or less comprehensively at the monthly meetings of the Branch, and also at the meetings of the various Sections.

The subject which I have presumed to treat would have been more worthily dealt with by some other member more capable than myself, and I have often wished that some older member of the profession would have felt the urge to address himself to it, and this feeling has been increasingly pressed upon me during the past seven or eight years of my membership of the Council.

I have chosen for my subject this evening "Medical Ethics" and I trust that although I shall speak boldly and plainly on my subject, I shall not in its treatment say anything but what is true and fair. It is well we should have a true perception of the real meaning of "ethics". Murray's Dictionary tells us that it is: "The science of morals, or a study of the principles defining a man's duty to his neighbour". That seems to me to be a fair description of what "ethics" really are in the abstract; the qualifying adjunct "medical" confines me to the consideration of those principles of ethics so far as they have to do with those who are members of the medical profession.

[1] Delivered at the annual meeting of the South Australian Branch of the British Medical Association on June 28, 1933.

During my connexion with the Council, it has been forcibly brought home to me that, although good progress has been made along the scientific side of the profession, we are undoubtedly retrogressing in the matter of medical ethics, and it seems to me that an address upon this subject is long overdue.

I would not, however, go so far as to say that medical ethics are honoured more in the breach than in the observance. All honour to those—and there are many—who do try to keep the high standard which as a profession it should maintain. This is an increasingly difficult task, for I fear that there are many within our ranks who give little thought to the ethical side of their professional duties, and the number of these, increasing as the years go on, makes one wonder why this should be the case.

Is it that insufficient knowledge is imparted to the student during the course of his training, or is it that we, as a body, are becoming too commercialized? Or is it that in these times of depression the medical man can focus his mind only on the one subject (outside the scientific side) of making money. As a matter of fact I think all three factors are responsible for the undoubted lack of medical ethics that exists in the profession today.

It has always been my opinion that the young medical graduate, on leaving the university at the end of his course, does so without a sufficient and efficient knowledge of the ethics of the profession, or of the correct treatment of his colleagues with whom he is about to enter into cooperation and friendly rivalry.

The newly fledged doctor has, at the time he has completed his course, had, I believe, two or three lectures only in medical ethics, and professional etiquette. It is impossible that two or three lectures given at the end of his course, and at the end of the year, are sufficient to impress upon the student the very great importance of the proper treatment that should be accorded by one medical man to another. In my opinion, such lectures should be given at least for the last three years of the student's course. I wish in this connexion to draw attention to the fact that house surgeons are often seriously in error, and commit many breaches of ethics by their practice of discussing with relatives of patients the diagnosis and treatment of the practitioner who happens to have sent the patient into hospital; for almost invariably the remarks, no doubt very often embellished, that the doctor's diagnosis was wrong and his treatment worse, come back to the medical attendant. The house surgeon cannot be too careful or too tactful in what he says to the relatives of patients, for he might by unguarded statements do much harm to a perfectly reputable and well informed medical practitioner. I am sure, if he thought of what the results of some of his unguarded statements might be, he would exercise a greater care and discretion in his utterances. But, as I have mentioned before, one can perhaps trace this failure on the part of the newly qualified man to his youth, and to the insufficient training he receives in medical ethics, and I suppose the failure is more or less excusable. But what of ourselves, the older members of the profession? We from our experience and our more mature years should not be open to the accusation that not only are we unable to enhance, but that we are doing much to lower the high standard that has been built up for us by the great men during the past century. I say most emphatically that we are open to such accusations, and would go even further and say that in my opinion the lack of medical ethics is not confined to one section of medical practitioners alone, but exists in some degree in all the ranks.

Why, I ask, is it that this should be the case? We cannot, I hope, put it down to the fact that fewer men of the right type are entering the profession, that we are in effect gradually becoming a body of men who think nothing of those principles to which I have referred, which guide men along what are generally esteemed to be the comely and cultured walks of life. If this be so, then there seems to me to be nothing so certain but that it will be a descent from the high standard which the medical profession has aimed at and attained.

Chauncy D. Leake, in tracing the historical development of medical ethics, notes two main paths of progression. The first is concerned with regulations imposed on physicians by outside, usually by the civil authority, and the second with regulations imposed upon medical practitioners by themselves, covering generally the question of fees and designed to protect at once the interests first of society and secondly of the individual practitioner. Many of the regulations made both before and after Christ were probably not initiated by physicians, but were rather laws to correct extortion and abuses practised by physicians.

Our first great law of the true medical ethics was that contained in the Oath and Law of Hippocrates. This apparently formed the standard of medical ethics until the publication of Percival's Code in 1803, and since the publication of this important document, up to the present time, most laws relating to medical practice have been instigated and established by responsible physicians and all standards of morality have been based on Percival's work.

Percival's "Medical Ethics" was a very comprehensive book written as it was well over a hundred years ago. The author discusses such subjects as: (i) professional conduct relative to hospitals or other medical charities, (ii) professional conduct in private or general practice, (iii) conduct of physicians towards apothecaries, (iv) professional duties in certain cases which require a knowledge of law.

It is not, however, the history of medical ethics that I wish to discuss tonight. I merely diverge a little from my subject to point out to you: (i) that medical ethics is a subject which has been in the minds of physicians for many a long century, (ii) that ever since the medical profession became

a cohesive body of men, it has been found necessary from time to time to formulate laws within the compass of which medical men could properly carry out their duties.

Lord Bacon in his "Common Law of Ethics" writes:

> A debtor to his profession from which as men of course do seek to receive countenance and profit so ought they of duty to endeavour themselves by way of amends to be a help and ornament thereunto. This is performed in some way by the honest and liberal practice of a profession when men shall carry a respect not to descend into any course that is corrupt and unworthy thereof: and preserve themselves free from the abuses wherewith the same profession is noted to be infected. But much more is this performed if a man be able to visit and strengthen the roots and foundation of the Science itself, thereby not only gracing it in reputation and dignity, but also amplifying it in profession and substance.

We can say that there are three main principles which can be regarded as the bases of medical ethics.

1. The relation of the medical practitioner towards his colleagues. Here the "golden rule" applies, "Whatsoever ye would that men should do unto you, even so do ye unto them".
2. The relation of the medical man to his patient.
3. The relation of the medical man to the State.

I put them in this order not because I think that a practitioner's behaviour towards his colleagues should be prior to his relation to his patients or even as a citizen of the State, but it is because it is his violation of the golden rule that is his greatest fault.

In his relation to his patient the practitioner will not err very often, at least not of direct intention. His patients supply his means of subsistence, and he knows full well that should he fail in his duty towards them, they can retaliate by dispensing with his services in the future.

In the relation of the practitioner to the State, he is bound to perform or to abstain from performing certain acts that are well known to be improper or injurious to the State, and the penalty for breach of such laws would act as a deterrent even if he wished to act contrarily.

As regards his relations to his colleagues, however, the case is very different. In this regard he is expected to follow a certain moral code, a code which, as I have said before, has been gradually built up along the centuries by some of the greatest and ablest men of their respective times in the medical world. Breach of this medical code very frequently can carry no other penalty than that of a feeling of distrust and disgust in the minds of his colleagues, and for all decent men this should be sufficient.

In all cases where a body of men are working on more or less definite lines to perform work more or less common to all of them, it has ever been found necessary to establish an association in which members of the profession may control other members, and to have a code of rules—apart altogether from restrictions laid down by the civic power—to keep its members from transgressing the rules of the association. In the generality of cases it has usually been found that moral pressure, the contempt of the orderly majority, has been sufficient to keep the unruly individual within the bounds of decent observance of the association's rules.

There is no greater subject for debate in medical ethics, just as there is no rule of medical ethics more frequently broken, than is that of advertising, or implied solicitation by advertisement. The medical profession has always set its face against the dishonour inflicted on the whole profession by such unworthy methods. That advertising has always been more or less practised, I think, gentlemen, you will agree. As far back as the eighteenth century, a satirical writer said of the famous Dr. Radcliffe: "On his first arrival he had half the porters in town to call for him at all the coffee houses, and public places, so that his name might be known" (Saundby).

The question naturally arises, why is it that we object to advertising? If the argument is that the advertiser is obtaining undue advantage over his colleagues, the obvious retort would be that if advertising were open to everybody, there would be nothing unfair about it.

Saundby in his "Medical Ethics" points out that the matter goes further than this. He points out that if a tradesman has an article to sell he may claim for that article any quality he pleases, regardless of the truth of his statements. In the case of most trade articles, purchasers are competent judges of the quality of the articles, and will purchase only those which they think are somewhere near the standard the advertiser claims for them. But there is a class of articles, such as patent foods, medicines *et cetera*, of whose value the average public are incompetent of judging. It is concerning these that most lies are told. If, then, the public are incompetent of judging the real value of these articles, how would they be able to discriminate between the claims of medical practitioners if they advertised? As a natural corollary, there would be very shortly a lowering of the standard of the profession, and we should have our ranks filled with sharp, shrewd business men, to the detriment of the true scientific man, and so to medical science as a whole.

In these days of many newspapers, picture shows, and loud speakers, the opportunities offering for advertising are manifold, and are, I am afraid, not infrequently used for advertisement.

The reporter, we know, is always looking for "copy", to seek whom he may devour, and sometimes he publishes over or under a doctor's name, if not an absolutely untrue statement, then very often a garbled or even reckless statement. In these cases I think the doctor who has been inveigled into this snare, is almost always more sinned against than sinning, but such interviews when published in the daily papers bring about a feeling of unrest among his brother members of the profession. During the year the newspapers were approached by Council and requested that doctors' names

should be left out of the accounts of these interviews. The Press representatives promised that they would do what was in their power to help the profession, but at the same time they pointed out that the "copy" would lose much of its value if they were not permitted to put the name of the doctor who was responsible for the contribution.

It would be a wise plan if almost always the doctor would refuse to be interviewed, but if he should decide to give "copy" to the papers, he should refrain from entering into discussion upon the subject of new treatment of disease. He should also be very careful in his casual remarks and should imperatively insist upon the suppression of his name. Another form of advertisement with which the Council dealt with in the daily papers, was that of doctors' names appearing in the "Birth Notices" column. This form of advertisement, as you will readily understand, is very difficult to prevent. The advertisement is inserted and paid for by the person interested and is purely a matter between him and the paper concerned. Because of this, the Council has authorized a notice to be sent out to you during the year, asking you to discourage in every way the very much increasing habit of doctors' names appearing in these notices. It is surely most unethical, to say the least of it, for a doctor to offer to pay for a notice such as this, provided that his name should be inserted.

Yet another way in which advertisement may be and is gained is by the giving of lectures on semi-medical subjects to a lay audience. If a request be made to a doctor to deliver a lecture on such a subject to the lay public he would, I think, be well advised to ask the Council for their opinion and approval. This is, as a rule, done, but not invariably. I know of such lectures being given to lodges by lodge surgeons. The obvious reason for a medical practitioner acting in this way could be only that of advertising himself among the lodges and gaining for himself patients, it may be from his colleagues who would not lend themselves to any such shady form of advertisement. It has always seemed to me to be totally unworthy and quite unnecessary for a doctor who happens to be attending a picture show to have his name thrown upon the screen asking him to call at the office. This is such a crude form of advertisement that it can be done only by someone entirely lacking in imagination.

Nor is it right for doctors present at large public gatherings to have their names blazoned abroad by means of loud speakers. There are those among us, I know, who can construe advertisement into some of the smallest trivialities. It may be that a medical man seeks municipal honours, or that he is unfortunate enough to have his name inserted in the newspapers because he has met with an accident to his motor car, or he has taken an aeroplane journey to consult with a colleague in the country, or even that his name appears as being present at a social gathering. I do not class myself among these objectors, but I do say that numerous means of advertising oneself are eagerly grasped by members of the profession. Such complaints as I have mentioned would be ridiculous were it not for the fact that there exists in the minds of some of us a feeling of jealousy, or it may be of antagonism, towards certain members, probably higher in the profession than others, while the complainant is only too anxious and eager to grasp the opportunity of pointing out to the other members of the profession how Dr. So-and-So invariably advertises himself. This is a failing which we should endeavour to combat, for it is a source of weakness in the profession.

It has long been considered unethical for medical men to accept honorary positions as medical officers to sporting bodies, such as racing clubs, football clubs and the like, and yet this rule is constantly being broken. Most, if not all, of the racing bodies of the metropolitan area do pay their medical officers an honorarium, but this rule, I believe, does not always obtain in the country. With football clubs the rule is being continually set at naught.

Another matter which the average practitioner is constantly coming in contact with is the position in which he stands towards a consultant.

It will be granted, I think, that when a patient is sent by a medical man to a consulting physician or surgeon for consultation, the consulting doctor should report to the patient's own medical adviser and return the patient to him. This procedure, I believe, is usually carried out, but I regret to say that this is not invariably the case. Sometimes the patient goes to the consultant never to return, nor is the doctor who sent the patient advised as to what has happened. Quite frequently a patient who is under treatment by his own doctor, for some obscure reason, will go to a physician or surgeon without the knowledge of the doctor who is treating him. He probably reasons that by so doing he will prevent collaboration of the two doctors and will obtain an unbiased opinion from the consultant. The question arises as to what is the correct form of procedure for the consultant in such cases. Should he treat the case as one sent to him by another doctor for his opinion or should he look upon the case as a legitimate one of his own?

The general consensus of opinion among those who have studied the ethics of the profession is "that a consultant is not bound to ask a patient coming to visit him the name of his medical attendant and may treat the case without reference to anyone provided that he can do so with justice to the patient. But he must not visit the patient for this purpose. If the patient requires treatment in bed the consultant should communicate with the regular attendant".

I mention this fact because such circumstances so frequently arise and it appears to me that the consultant is quite right in his actions up to a certain point. Not uncommonly, after this point is reached, he is frequently wrong. He sends the patient into hospital or to bed at the patient's own house and continues his treatment there. This

quite naturally leads to ill feeling and perhaps complaints from the usual medical attendant.

Yet another class of case is that in which a patient is taken ill while at his work. A doctor near by is called in and orders him or her to a private hospital where he continues to treat the patient until recovery or the reverse happens. These cases are really emergency cases and should whenever possible be treated as such. That is, the patients should be given relief for the time and returned to their own regular doctor.

Of course I realize that sometimes such procedure is not possible, but it is not uncommon for a patient to stroll into his doctor's consulting room and inform him that he has been operated upon for appendicitis or gall-stones or some such illness; and this is the first intimation that the regular medical adviser has had. It is rather amazing to find how few doctors realize that emergency cases are not correctly theirs. A little while ago a medical man volunteered the information that in cases of accident brought to his surgery he continued to treat the patients until they had recovered. He apparently had no idea that he was doing something unethical. He did not appreciate the fact that such patients as these came to him only from force of circumstances and they would have preferred, if they could, to have had their own medical man. Allied to this question is one that not infrequently comes before Council, and that is the question of the position of insurance referees, and of referees in the employ of large industrial bodies in regard to practitioners. During the year a copy of rules governing the procedure in such cases has been sent to you.

The patient in these case is forced to go to the medical referee. Failure to do so would mean either dismissal from employment or loss of compensation. There are doctors, however, who hold the position of referee, who do not hesitate to retain the patient, and who continue to do so either until he has recovered or until the company decides to pay some lump compensation. The patient may or may not be a private or lodge patient of another medical man. The principle in either case is exactly the same and the end result is that the referee has filched a patient from his usual attendant, whom the patient had at no time wished to leave. A question that is frequently coming before the Council is: "Is it ethical for a practitioner to commence practice in a town where another member of the profession is also practising?" It has been ruled on many occasions that such a thing is permissible. A doctor can commence practising wherever he chooses. But is it, let me ask, always wise?

Almost invariably the results of the invasion of a new medical man into the town where a colleague is already practising leads to enmity between the two, the division of the inhabitants into two parties, string pulling to obtain any appointments that may be available, and very frequently the starvation of one or other of the medical men. I think a doctor who is considering entering into competition in a small district would be well advised to consider seriously the effects of his decision and the troubles, and even danger that most assuredly lie ahead of him.

Many years ago it was, so far as I am able to judge, the usual practice of a dentist to draw attention to the fact that any work done by another dentist was invariably bad. Whether that practice exists today I do not know, but I often think that we ourselves are retrogressing towards the practice that existed with dentists in those days. Undoubtedly there are medical practitioners within our ranks, and many of them, who should know better, who do not hesitate to do and to say what they can to the detriment of another doctor. If they do not actually tell the patient that it is lucky for him that he has come along just in time to avoid disastrous results, they often infer the same thing which is probably worse.

When one considers the great progress that has been made in medical science during the past forty or fifty years, it cannot be expected that the average general practitioner can attain perfection in all the branches of medicine and surgery and must on many occasions depend upon the specialists of the various branches for consideration of the cases about which he is unable to come to a clear conclusion. This he realizes and sends those cases to a consultant for his opinion. It is, therefore, abominable on the part of the consultant to say anything to the detriment of the doctor concerning the treatment of the patient to whom he has ministered and it is the acme of dishonour to take the patient and treat the case as if it were his own. In doing so, he in the one case lays the colleague open perhaps to the action of malpraxis by the patient and in the other he filches a patient.

I have brought to your notice this evening many forms of breaches of medical ethics. I could, if I chose, give you many more. I have not, for instance, mentioned the practice that is becoming more and more apparent and that is that of "undercutting".

If it be granted that owing to present financial conditions medical fees should be on a lower basis than they have been in the past, at least let this reduction be uniform. It is not right to my mind that fees (and here I refer more particularly to general practitioners) should be lower in one district than in another, and I feel this point more strongly still when individual doctors in a particular district lower their fees in an attempt to undercut those of their colleagues.

Nor have I mentioned the practice of dichotomy that exists amongst us. This practice exposes members to the charge of arranging consultations and operations for their own personal interests when such consultations and operations are not really necessary. It also, by these methods, throws suspicion upon the consultant or operator of charging greater fees than are warranted, so that he can recompense himself and obtain what in the normal way would be for him a usual fee.

I have not referred to the increasing habit which unfortunately, of later years, has come into vogue, that of "touting". It seems almost incredible that a medical man could stoop to the practice of "touting" for patients, yet amongst a certain class it is certainly done. Sometimes this canvassing for patients is done personally and sometimes it is done through the medium of others such as chemists, midwives or even nursing homes and hospitals. I have no words at my command which can adequately express my views upon such a practice as this, and I hope you all will set your face against it.

I trust that I am not going outside my province if I make a few remarks on the influence which we older members of the profession may exercise on those whose future lies still open and more or less undeveloped. May we not as fellow members of a noble, and of what is in many ways a self-sacrificing, profession attempt to keep practice and profit in their proper places.

We can do this by emphasizing in our discharge of our duties their relative importance. Must we not feel in our higher moments that we must do all we can to enhance the profession's honour and to maintain its glory. Compared with this, of what value are the shekels that we put into our pockets, even in so avaricious an age as that in which we are living today?

"Il faut vivre"; it is true we must live and must provide sustenance for ourselves and for those belonging to us, but to be forever thinking how rapidly we may add to our bank balance—if we have one—and to do this by breaking down our moral codes does not seem to me to be in tune with the duty which we owe to the profession of which we confess ourselves to being proud of being members.

"Put money in thy purse" says Iago in the play, but let us be sure the money is untainted with dishonour to those who are fellow members of the profession.

"Pecunia non olet"—money does not smell; quite so, if it be fairly and honourably acquired, if that be not the case, then "it stinks to heaven" and pollutes the atmosphere in which we live.

> Worth makes the man and want of it fellow
> The rest is all but leather and prunella.

It is time that I brought these crude remarks of mine to a conclusion, and I thank you for the patience with which you have listened to me. I should like before I close to offer a few remarks suggested by the topic upon which I have been speaking.

There is, or there should be, a wide difference between ordinary trades and what are called the liberal professions. Has it ever occurred to you to ask the reasons for this wide difference? Trade or barter or by whatever name it shall be called implies an intention of making pecuniary profit. The original idea was that the liberal professions, in their origin, were free. The lawyer served his client, and, the case over, the client put what money he could afford into the bag which was hung over the lawyer's shoulder. The physician was not supposed to be paid on "the knocker", but his fee was left for him on a convenient table, and the true-hearted priest was paid by alms.

While distinctions of these high offices show the underlying spirit which ought to mark all the liberal professions, it is true that lawyers, doctors and some clergy make money. But what I want to emphasize is the fact that at their first origin and early development they were free servants of a troubled, pained and suffering humanity.

It would be a pity, a thousand pities, if this noble ideal were altogether lost. At any rate, let us do our best to retain as much of it as still remains. For is it not true that in our profession breaches of medical ethics must lower the standard of the profession? And will not the result of any such abasement be that the entrance of a wrong class of man will be made easier? And will not this again spell the lowering of the standard of scientific work?

Am I wrong in saying that the tendency of many of those who contemplate entering the profession today is to discard all thoughts of the traditions of the profession and its benign and blessed work, and to look upon it, and its work, merely from a baser and commercial standpoint?

The future of the profession lies in the hands of the younger generation, and let it be remembered that the example for the younger must be set by their elder brethren.

Let me close with an old but very short story. A young lawyer first entering his father's profession asked his parent for advice as to his future career. He replied: "My son, the first thing that you have to do is 'to get on', the second is 'to get honour' and the third is 'to get honest'." Reverse them, let me say, and you will have a good prescription for a young medical man.

> Ill fares the land [or any association of men] to
> hastening ills a prey,
> Where wealth accumulates, and men decay.

SIMULATION OF VASCULAR DISEASE OF THE GENICULO-CALCARINE PATHWAY BY CEREBRAL TUMOUR.

By KENNETH B. NOAD, M.B. (Sydney), M.R.C.P. (London),
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HEMIANOPIA occurring in the middle-aged is encountered occasionally in ophthalmological practice, and when associated with papillœdema or other obvious phenomena of cerebral damage, such as weakness of opposite face, arm and leg, and contralateral disturbances of sensation, is usually recognized as having a serious pathological basis.

In the absence of papillœdema or neurological signs, vascular disease with thrombosis of the

calcarine branch of the posterior cerebral artery is most often thought to be the cause, as this is said to be much the commonest basis of occipital hemianopia.

However, in the middle-aged, cases without papilloedema are encountered in which hemianopia occurs as an isolated symptom or associated with equivocal neurological signs. In such cases an accurate diagnosis, either anatomical or pathological, is difficult.

Cairns[1] has written recently that "experience has served to emphasize the fact anew that homonomous hemianopia should usually be regarded as indicating merely the side rather than the site of the lesion", and we might add "or the pathology" in many cases.

This paper contains an account of two cases which simulated vascular disease, but were due to tumour. Some of the pathological lesions giving rise to hemianopia are considered and an attempt has been made, from the cases described, to discover what symptoms and signs might have been helpful in establishing a correct diagnosis.

Anatomy.

A consideration of the anatomical features and blood supply of the geniculo-calcarine radiation is germane to this discussion.

Each optic tract terminates in the superior colliculus and pulvinar of thalamus, but chiefly in the external geniculate body of the same side, from which a fresh relay of fibres passes out into the posterior limb of the internal capsule, behind the fibres of common sensation on their last relay to the cortex, and internal to the auditory radiation.

From this situation dorsal and lateral groups of fibres pass back directly to the occipital lobe, rounding the lateral wall of the posterior horn of the lateral ventricle in their course. According to Meyer,[2] the ventral fibres form a detour by bending forwards deeply in the substance of the temporal lobe round the anterior horn of the lateral ventricle and then, turning sharply backwards along the inferior aspect of the ventricle, to join the dorsal fibres in the occipital lobe.

Holmes and Lister[3] and Roenne[4] suggest that there may be an anatomical interval between these sets of fibres which contains the macular bundle.

The visual fibres finally impinge on that area of the occipital cortex distinguished by Gennari's line. This roughly comprises the area above and below the calcarine fissure and within its lips. It also extends round the occipital pole on to its lateral surface.

Blood Supply.

The main supply of the geniculo-calcarine pathway is from the posterior cerebral artery, especially its calcarine branch. Posteriorly the marrow of the occipital lobe also receives penetrating twigs from the middle cerebral artery.

The *area striata* or visual cortex is also nourished principally by the calcarine artery. At the pole a pial anastomosis is formed with twigs from the middle cerebral artery, so that macular vision, which is here represented, has a double source of supply. It is consequently spared in thrombotic closure of the calcarine artery with the production of a characteristic perimetric field defect—hemianopia with sparing of the fixation area.

Pathology.

Vascular Disease.—According to Traquair,[5] the commonest cause of occipital hemianopia is vascular closure due to thrombosis or embolism with softening of the lobe. Hæmorrhage is a less frequent agent.

These may affect the posterior cerebral artery itself. Its fellow of the opposite side is also not infrequently involved with the production of bilateral hemianopia. Perhaps a minute central field may remain, corresponding to the bilateral sparing of the fixation area. In not a few cases, however, complete blindness is the result.

More commonly it is the calcarine branch of the posterior cerebral artery which is affected. The symptoms of its occlusion are often so slight as to escape the patient's notice until the apparent incursions of passers-by on his blind side reveal the condition.

The field defects so produced may remain stationary and absolute, or show some degree of recovery, but their chief characteristic is the escape of the fixation area.

Neoplasm.—Tumour pressure, in Traquair's opinion, is a far less common cause than vascular disease. The neoplasms occurring in the hemispheres at the middle period of life may be: (i) Glioblastoma, rapidly growing, radio-insensitive, and recurring rapidly after removal. Operative interference with this type of growth, however, usually provokes a rapidly spreading cerebral œdema and death. (ii) Astrocytoma, slow growing, relatively benign and frequently cystic. Drainage of the cyst and removal of the mural nodule usually present frequently allow some years of normal life to be enjoyed. (iii) Oligodendroglioma, rare and the most slowly growing cerebral tumour of adults.

Abscess.—Uhthoff found homonomous hemianopia in 9% of patients with cerebral abscess. More than half of the abscesses occurred in the occipital lobe.

Migraine.—Migraine may give rise to temporary hemianopia, which is liable to recurrence in subsequent attacks. Rarely it may be permanent and complete from the first attack.

Trauma.—Trauma is an infrequent cause in times of peace. It is commonly associated with involvement of the fixation area.

Case Reports.

CASE I.—The patient was a male, aged fifty-six years, admitted to Sydney Hospital in August, 1925. His history was one of progressive deafness in the right ear, which had begun two and a half years previously and had become complete. The left ear meanwhile had also become involved. He complained of a ticking noise in the right ear and ringing sounds in both ears.

He had been giddy at times and had vomited occasionally. He had had headaches during the past two months, worse in the morning, and tending to wear off during the day. His vision had been failing during the past six weeks. At first he thought he could see a shadow of someone standing on his left. Subsequently he noticed that he walked into people on his left side. He had seen a play of colours before both eyes, and sometimes things seemed to have a "different appearance", for example, people's features would appear broader than normal, "vehicles would seem to swing round to the left".

He had been short of breath for some years, and two and a half years previously had been in Sydney Hospital with a failing heart.

On examination he was intelligent, cooperative, and showed no sign of mental impairment.

On August 7 he was found to have a homonomous hemianopia, and the ophthalmologist (Dr. R. B. North) reported that the fundi were normal. His pupils were equal and reacted to light and accommodation. There was well marked horizontal nystagmus on looking to the left, less marked on looking to the right. He was unable to hear a watch ticking when it was placed on ears or skull.

There was some increase of tone on the left side, with increase of deep reflexes and a doubtful extensor response on the left side.

Some impairment of tactile sensation and discrimination, passive position and passive movement existed in the left arm and leg. There was no adiadochokinesia.

His gait was normal and showed no ataxia. Romberg's sign was absent. The examination of his cardio-vascular system revealed that his pulse was 75, regular, collapsing in type. There was a mitral systolic murmur conducted into the axilla and an aortic diastolic murmur conducted down the left side of the sternum. The blood pressure was not recorded.

The patient's condition rapidly deteriorated. He suffered a good deal from headache and vomiting, and developed some right-sided incoordination and adiadochokinesia, and a tendency to fall to the left on walking.

The cerebro-spinal fluid was clear, but under increased pressure, and it contained an excess of globulin. Together with the blood it failed to react to the Wassermann test.

An X ray examination of the skull revealed marked irregularity of the *sella turcica*.

The patient died on September 8, 1925, in coma, with a rising temperature. The notes contain no further reference to the condition of the optic disks.

At autopsy a large tumour was found in the right occipital lobe, extending forwards towards the thalamic region. Part of the tumour was cystic and contained straw-coloured fluid.

I can find no reference to the histology of the tumour, but it was almost certainly an astrocytoma.

The heart showed no pericarditis, much thickening of the mitral valve, but no lesion of the aortic valve.

In this case the relative importance of the various symptoms and signs was not correctly assessed during life. A distinguished consultant expressed the consensus of opinion when he wrote, on August 17, 1925: "This is a case of gross intracranial disease, possibly of diffuse degenerative nature rather than pressure of tumour. The cardio-vascular system also shows degenerative change."

On reviewing this case at the present time, neoplasm would seem to be much the likelier diagnosis. Too much reliance was apparently placed upon the absence of papillœdema at the time of the recorded examination. That the disks remained normal there is no note. It is a fair assumption that examination of the fundi during the days of his rapid decline would have revealed blurring of the disk edges or even a more marked degree of papillœdema.

The association of hemianopia with other sensori-motor phenomena indicated that the visual fibres were involved in the anterior part of their course before they had parted company with the internal capsule. This syndrome might have been produced by a vascular lesion, but in this instance the onset of severe headache and vomiting, the visual hallucinations in the form of flashes of light, the development of homolateral cerebellar signs, from pressure downwards through the tentorium on the lateral lobe of the cerebellum on the same side, indicate new growth as being the likelier diagnosis.

The presence of cardio-vascular degeneration and the absence of papillœdema provided a real difficulty in arriving at a correct diagnosis. This is evident from the erroneous conclusions arrived at by the clinicians who saw the patient.

The tardy recognition by the patient of the presence of a field defect (it was unnoticed till less than three months before his death), to my mind, constituted a remarkable feature of this case. With a lesion the size of that found *post mortem* some defect must have existed for a considerable time before it was noticed, but an homonomous hemianopia had developed before the patient was aware of any limitation of his field of vision. This fact, taken in association with the somewhat equivocal symptoms of the patient on his admission to hospital, might have seemed to justify a tentative diagnosis of vascular disease, but subsequent developments should have excluded such a conclusion.

CASE II.—T.N., a male, aged fifty-two years, was admitted to Sydney Hospital on June 21, 1932.

The history was one of gradual psychological change. He had felt out of sorts since 1927. Formerly a happy and jovial man, he was now subject to "cranky, irritable turns for no reason". He would become excitable and want to fight his sons on the slightest provocation. He had fretted a great deal over his daughter's untimely death in 1929.

He had recently begun to complain of pain in the occipital region, of dull, aching character, with sharp stabbing exacerbations in the frontal regions, coming on chiefly in the morning. This had become worse since the beginning of June, 1932.

He had vomited occasionally during the past few years, and on three occasions during June, 1932.

He had constant noises in the head, "like water dripping on tin".

On one occasion he had seen flashes of light before his eyes, and for at least the past twelve months had been unable to see anything on his left side. This had been noticed quite suddenly in the street one day, when he had bumped into passers-by on that side.

His memory had become somewhat impaired during the past two years.

Physical examination revealed that he was mentally rather dull. His speech was slow. His cooperation was slow, but fairly efficient.

His cranial nerves were intact, the disks were normal, and his visual fields showed a left homonomous hemianopia, with sparing of the fixation area. The vision in the right eye was $^6/_{36}$ and in the left $^6/_{60}$ (see Figure I).

The remainder of the examination revealed no neurological abnormality.

Examination of his cardio-vascular system showed a palpable and tortuous radial vessel. The pulse was 70 in rate and regular. His heart dulness was increased slightly to the left, but the sounds were clear.

X ray examination revealed some cardiac hypertrophy and electrocardiography demonstrated a left ventricular preponderance, with a pulse rate of 96 per minute.

There was some tenderness over the left antrum, which was also dull on X ray examination. Proof puncture revealed foul pus, and a left radical antrostomy was done on July 7, 1932.

The cerebro-spinal fluid was clear, under increased pressure, and contained 15 mononuclear cells per cubic millimetre, but no other abnormality.

Neither blood nor cerebro-spinal fluid reacted to the Wassermann test. An X ray examination of the skull revealed no abnormality. A well marked pineal shadow was visible, central in position.

Following the antrostomy there was some improvement in his general condition, and he seemed brighter, but his headaches returned with increased severity, finally becoming absolutely uncontrolled by either 50% magnesium sulphate given rectally or by intravenously given sodium chloride.

His mental condition also deteriorated and finally sphincteric incontinence developed.

On July 28, 1932, commencing papilloedema was observed. Decompression was performed on the following day. The patient died on July 31, 1932.

At autopsy a tumour was found in the white matter of the right occipital lobe. Its situation can be seen in the accompanying photograph (Figure II).

In this case a provisional diagnosis was made of cerebral vascular disease, with thrombosis of the calcarine branch of the right posterior cerebral artery.

This seemed a reasonable assumption in view of the long history of increasing psychic change, the presence of vascular degeneration, and a characteristic visual field with sparing of the fixation area.

The following extract is taken from Traquair's book, "Clinical Perimetry":

In occipital hemianopias of vascular origin sparing of the fixation area is almost constant, and is more extensive and complete than in subgeniculate hemianopia. The spared area is usually less than five degrees in extent, but may be more, and has been found occasionally to extend upwards or downwards or in both directions along the vertical meridian for some distance, merging into the overshot field, which attains its fullest expression in exceptional cases where the sparing extends to the periphery both above and below.

If the accompanying perimetric chart is studied, it will be seen how closely this description fits the type of field obtained in this patient.

As in the first case, the field defect formed a remarkable feature. The lesion had assumed a hemianopic nature before it was noticed quite suddenly. It subsequently persisted unchanged for one year. These facts also influenced the diagnosis in favour of a vascular accident. The complete absence of signs of organic nervous disease seemed also to fortify this view.

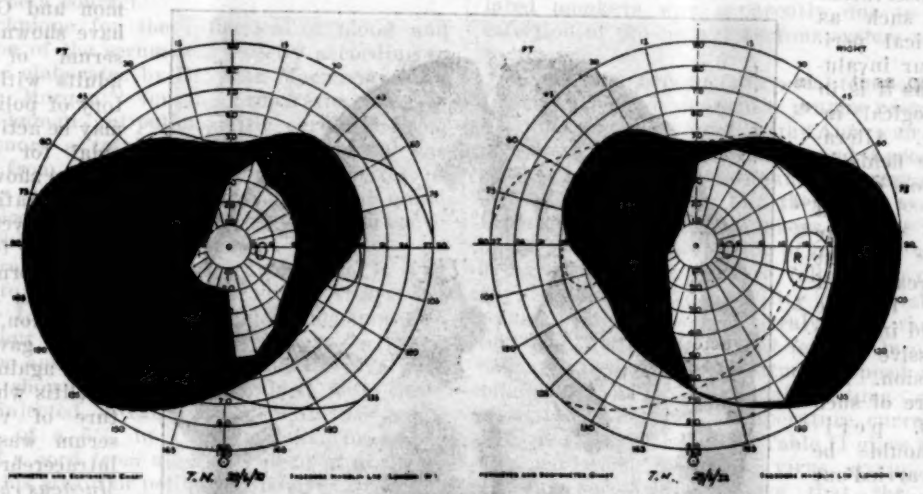

FIGURE I.

Showing left homonomous hemianopia with sparing of fixation area and that part of field adjacent to vertical meridian above and below fixation point.

On reviewing the history, there is one symptom to which, perhaps, due importance was not attached. This was visual hallucination in the form of flashes of light, which had occurred on one occasion. This is a common occurrence in tumour of the geniculo-calcarine pathway, or of the cortex itself, and rarely occurs in vascular lesions. This symptom definitely favoured a neoplastic basis of the condition, and had it not been of such a transitory nature, more notice would have been taken of it.

It is interesting to note that in these two cases there was a marked similarity of the subjective auditory phenomena.

Conclusion.

Two cases of cerebral tumour are described which during the life of the patients were diagnosed as cerebral vascular disease. These cases I have personally encountered, and my object in reviewing them has been to point out that in homonomous hemianopia occurring in the middle-aged a guarded prognosis should be given, as in many features new growth may closely simulate vascular disease.

In view of the frequency of cerebral neoplasm, all such cases encountered in ophthalmological clinics should be submitted to careful neurological examination. If there is a history of visual hallucination or if sensorimotor phenomena are present, these should be viewed with suspicion. Cerebellar phenomena on the same side as the occipital lesion would almost certainly indicate new growth.

These cases illustrate the futility of relying on the absence of papillœdema in excluding neoplasm of the occipital lobe, for in growths of this region it commonly appears in the terminal stages only.

In the observation of cases such as these, clinical perimetry is our invaluable help, as it is in all neurological investigation. Alteration of the fields in the direction of improvement would favour a vascular pathology, whilst any increase of limitation in the fields would indicate a progressive destructive lesion. But to be aware of such alterations, perimetry should be regularly carried out at least once a week, instead of the single perfunctory examination which is so frequently the case.

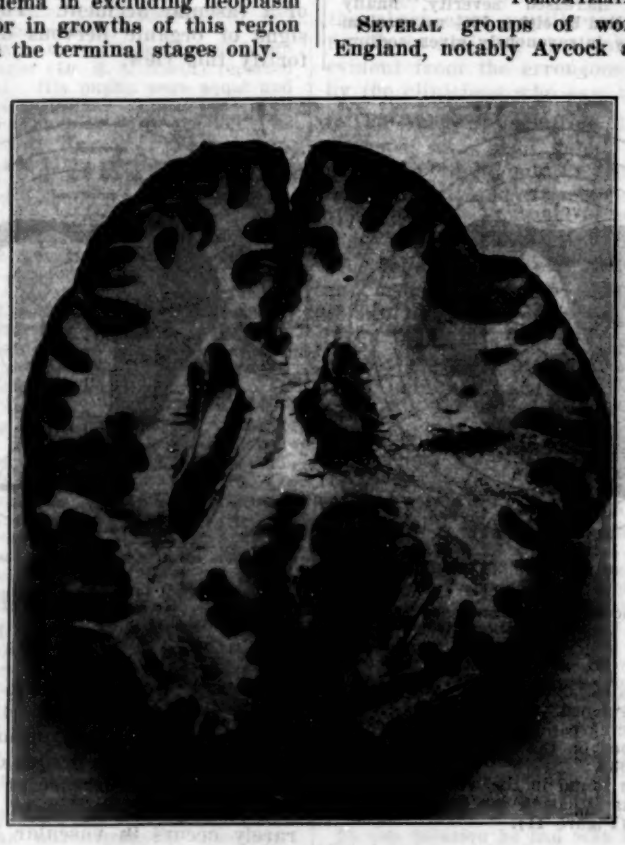

FIGURE II.

Cut surface of brain showing tumour in right occipital lobe. Marked *hernia cerebri* is present as a result of decompression. The hæmorrhagic area in the right *centrum semiovale* followed exploratory puncture of the hemisphere at time of operation.

Acknowledgements.

I have to thank Dr. H. C. Adams for permission to report Case II, and Dr. Wilfred Evans for Case I. I have also to thank Miss V. E. Jones, of the Pathological Department, Sydney Hospital, for the excellent photographs accompanying this article.

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SOME EXPERIMENTAL WORK ON POLIOMYELITIS.

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PART I.

A COMPARISON OF THE NEUTRALIZING POWER OF CONVALESCENT AND NORMAL SERUM FOR POLIOMYELITIS VIRUS.

SEVERAL groups of workers in America and England, notably Aycock and Kramer,[1][2][3] and Shaughnessy, Harmon and Gordon,[4] have shown that the serum of normal adults with no history of poliomyelitis may be actively virucidal for poliomyelitis as shown by the neutralization test. Thus Aycock and Kramer[3] found that of 41 normal adult sera from an urban population, 36, or 87·8%, gave protection against poliomyelitis when a mixture of virus and serum was injected intracerebrally into *Macacus rhesus* monkeys. Brodie,[5] working on sera from 29 normal adults (13 contacts and 16 non-contacts), concluded that "the pooled sera of known contacts and non-contacts were respectively a little above and a ltitle below half the strength of the average obtained from pooled convalescent serum".

From the foregoing results it seemed likely that normal adult serum would be of therapeutic value, and the main object of the present investigation was to determine the virucidal power of pooled normal serum from the adult population of the City of Melbourne. In the event of the serum being sufficiently potent, it was considered possible that it might eventually replace the convalescent human serum for therapeutic purposes or at least serve as a valuable adjunct in the event of a sudden demand for large quantities of serum. Dr. John Dale, Chairman of the Poliomyelitis Committee of

Victoria, was instrumental in launching a movement to obtain a supply of such serum, and it was arranged that a number of members of the permanent staff of the Town Hall, Melbourne, should act as donors. The group comprised thirty-two members, the ages ranging from nineteen to forty-six years. All were in constant employment and in a good state of general health, and were therefore considered to be ideal subjects for such a supply of serum. As poliomyelitis had been prevalent in the community during the years 1929, 1930 and 1931, it seemed reasonable to suppose that at least a proportion of these donors would have been subclinically immunized, although none was definitely known to be a contact.

The technique for the collection of blood and preparation of the serum was exactly according to the method elaborated by Dr. Jean Macnamara and Dr. F. G. Morgan[6] for the preparation of convalescent human antipoliomyelitic serum. From the 32 donors a total of 13 litres of blood was obtained, from which 5,380 cubic centimetres of serum was finally prepared, the individual quantities of blood varying from 180 to 600 cubic centimetres. The serum was prepared in 1932.

As a preliminary to testing the serum it was necessary to accumulate stocks of virus. At first the local virus LV, used by Burnet and Macnamara,[7] was used, but the strain appeared to have decreased in virulence and produced only moderate paralysis.

Table I shows the results obtained with three monkeys injected intracerebrally with 0·5 cubic centimetre of a 5% to 10% emulsion in saline solution of a cord from a monkey dead after infection with this strain of poliomyelitis virus. In each case paralysis was confined to the lower limbs.

TABLE I.
Results Obtained with Local Strain LV.

Monkey.	Day of First Paralysis.	Notes.	Outcome.
K2	9-12	Mild paralysis with slow onset. Legs partially paralysed. No other paralysis.	Recovered.
K7	10	Legs completely paralysed. No other paralysis.	Killed on twelfth day. Cord passaged to K9.
K9	10	Mild paralysis. Legs paralysed below knee only. No other paralysis.	Recovered.

Since this virus appeared to be of only moderate activity, cords from five fatal cases of poliomyelitis were tested. Intracerebral inoculation into monkeys of four cords produced no symptoms of paralysis, but the fifth cord contained an active strain of virus.

This cord was obtained from a child of seventeen months at the Children's Hospital, Melbourne. The patient was extensively paralysed in limbs and trunk muscles and died of respiratory failure on the third day of the illness. After ten months the injection of an emulsion of this cord caused paralysis on the ninth day after inoculation, the monkey being almost completely paralysed by the tenth day, when it was killed and the cord was removed. Passage through monkeys enhanced the virulence somewhat, and in subsequent experiments animals inoculated intracerebrally were completely paralysed by the eighth or ninth day at the latest. Usually the monkey was chloroformed when prostrate, the cord being removed aseptically, but if the disease was allowed to run its course, the infection was always fatal. In each instance a *post mortem* examination revealed no pathological lesions other than those consistent with poliomyelitis. One point of interest was the presence in each animal examined of a gross glycosuria of up to 6% of sugar in specimens obtained from the bladder. A small control series of specimens from eight normal monkeys showed a faint trace of sugar in two animals, while the remainder were sugar-free. The glycosuria in inoculated monkeys was apparently due to the acute infection of the central nervous system rather than to trauma.

In our later experiments rectal temperatures were taken and the temperature graphs resemble those of monkeys inoculated with American virus, as described by Kramer, Hendrie and Aycock.[8] The normal temperature was found to be approximately 39·3° C. (102·8° F.), but there was considerable variation in individual monkeys. In the inoculated monkeys the first indication of infection was always a sharp rise in temperature, followed in one or two days by anorexia, irritability or excitability and tremor, with subsequent paralysis on the third or fourth day after the initial fever. Once paralysis had appeared, the temperature dropped rapidly to subnormal as the animal became completely paralysed. A typical temperature curve (monkey K15) is given (Figure I). Table II gives the results obtained when this local virus strain was used without serum and shows that the strain is uniformly highly virulent.

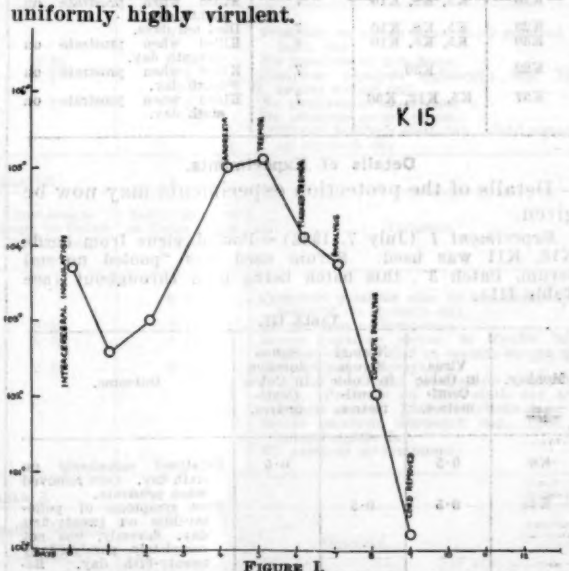

FIGURE I.
Temperature curve of monkey K15 inoculated with Australian LVii virus.

It was therefore decided to use this strain (LVii) for testing the neutralizing power of convalescent human serum and of pooled normal adult serum.

The technique used in the serum neutralization experiments was that described by Burnet and Macnamara.[7] A fresh emulsion of pooled cords was made in normal saline solution to about 5% strength. Mixtures with serum were then made as required, and after fifteen to twenty minutes at room temperature were left overnight in the ice chest before being injected intracerebrally.

The injections were made about 1·2 centimetres deep in brain substance through the intact dura, which was exposed in the right frontal region by a trephine or in some later experiments by a drill. Since some of the injected material usually escaped along the track of needle puncture when the needle was withdrawn, the actual bulk of injection did not seem to be important. We used a volume of 1·0 cubic centimetre except in two experiments, where there was a large proportion of serum in the virus-serum mixture and where slightly larger volumes were injected.

TABLE II.

Results Obtained with Local Strain LVii.

Monkey.	Cord used for Inoculation.	Day of First Paralysis.	Notes.
K10	Original cord from fatal case in child.	9	Killed when prostrate on tenth day.
K11	K10	6	Killed when prostrate on seventh day.
K12	K11	6	Killed when almost completely paralysed on seventh day.
K8	K10, K11	6	Killed when prostrate on sixth day.
K5	K10, K11	8	Killed when prostrate on ninth day.
K16	K10, K11	9	Died eleven days.
K15	K5, K8, K10	7	Killed when prostrate on ninth day.
K20	K5, K8, K10	7	Killed when prostrate on eighth day.
K23	K5, K8, K10	7	Died ten days.
K30	K5, K8, K10	8	Killed when prostrate on eleventh day.
K22	K30	7	Killed when prostrate on tenth day.
K37	K5, K12, K30	7	Killed when prostrate on ninth day.

Details of Experiments.

Details of the protection experiments may now be given.

Experiment I (July 7, 1932).—Pooled virus from cords K10, K11 was used. Serum used was "pooled normal serum, Batch 3", this batch being used throughout (see Table III).

TABLE III.

Monkey.	Virus in Cubic Centimetres.	Normal Serum in Cubic Centimetres.	Saline Solution in Cubic Centimetres.	Outcome.
K8	0·5		0·5	Paralysed completely on sixth day. Cord removed when prostrate.
K13	0·5	0·5		First symptoms of poliomyelitis on twenty-first day. Severely, but not completely, paralysed by twenty-fifth day. Recovered.

The effect of normal serum in this case has been to lengthen the incubation period considerably and to modify the disease to some extent.

Experiment II (August 8, 1932).—Cords used were again K10, K11 (Table IV).

TABLE IV.

Monkey.	Virus in Cubic Centimetres.	Normal Serum in Cubic Centimetres.	Saline Solution in Cubic Centimetres.	Outcome.
K5	0·5		0·5	Paralysed completely on eighth day. Cord removed on ninth day.
K14	0·5	0·2	0·3	Moderate paralysis gradually developing from ninth to twelfth day. Recovered.
K15	0·5	0·3	0·2	No paralysis or symptoms.

This experiment, from which it appears that 0·3 cubic centimetre of normal serum can protect against 0·5 cubic centimetre of virus does not support the first or later experiments. Probably the pieces of cord from which the emulsion was made contained less virus than usual. In later experiments the neutralizing power of convalescent and pooled normal sera was compared. Convalescent serum was taken from the stock held for therapeutic purposes.

Experiment III (September 6, 1932).—Cords used were again K10, K11. The convalescent serum was recently prepared (Batch 54, May, 1932) (Table V).

Here 0·25 cubic centimetre of convalescent serum completely protects against 0·5 cubic centimetre of virus, while 0·5 cubic centimetre of pooled normal serum protects almost completely, but not entirely.

Experiment IV (October 24, 1932).—Cords used were K5, K8, K10. Convalescent serum was Batch 45 (Group I, January, 1931) (Table VI).

From this experiment it appears that 0·1 cubic centimetre of convalescent serum is not sufficient to neutralize 0·5 cubic centimetre of virus, but that 0·5 cubic centimetre of pooled normal serum completely protects against the same volume of virus.

Experiment V (November 16, 1932).—Cords used were K5, K8, K10. Convalescent serum was again Batch 45 (Table VII).

Apparently 0·2 cubic centimetre of convalescent serum is unable to protect against 0·5 cubic centimetre of virus emulsion. With the two normal serum monkeys the question of individual variation arises. In this instance the difference in size of the monkeys was possibly a factor, since K26, which showed no symptoms, was much larger than K27, which received the same injection and was completely paralysed as a result.

Experiment VI (December 7, 1932).—Cords used were K5, K8, K10. Convalescent serum used was Batch 47 (Table VIII).

As these results were not altogether consistent with those previously obtained, a further experiment was carried out with the hope of getting absolute protection with both sera.

Experiment VII (February 10, 1933).—Cords used were K5, K8, K10. Convalescent serum used was Batch 54 (Table IX).

Experiment VIII (May 5, 1933).—As a final test of the pooled normal serum a mixture of serum and virus was made in the usual way, so that it contained 0·9 cubic centimetre of serum to each 0·5 cubic centimetre of 5% virus emulsion. The details of the experiment are shown in Table X. Cords used were K5, K12, K30.

TABLE V.

Monkey.	Virus in Cubic Centimetres.	Normal Serum in Cubic Centimetres.	Convalescent Serum in Cubic Centimetres.	Saline Solution in Cubic Centimetres.	Outcome.
K16	0·5			0·5	Paralysis nine to ten days. Died on eleventh day.
K17	0·5	0·5			Very mild paralysis confined to one group of shoulder muscles on tenth day. Recovered.
K18	0·5		0·25	0·25	No paralysis or symptoms.

TABLE VI.

Monkey.	Virus in Cubic Centimetres.	Normal Serum in Cubic Centimetres.	Convalescent Serum in Cubic Centimetres.	Saline Solution in Cubic Centimetres.	Outcome.
K20	0·5			0·5	Prostrate on eighth day. Cord removed.
K19	0·5		0·1	0·4	Completely paralysed on eleventh day. Died on twelfth day.
K21	0·5		0·1	0·4	Incomplete left facial paralysis on thirteenth day. Recovered.
K22	0·5	0·5			No paralysis or symptoms.
K23	0·5	0·5			No paralysis or symptoms.

TABLE VII.

Monkey.	Weight in Kilograms.	Virus in Cubic Centimetres.	Normal Serum in Cubic Centimetres.	Convalescent Serum in Cubic Centimetres.	Saline Solution in Cubic Centimetres.	Outcome.
K15	3·17	0·5			0·5	Prostrate on eighth day. Cord removed on ninth day.
K24	2·41	0·5		0·2	0·3	Complete paralysis ninth day. Killed when moribund on fifteenth day.
K25	2·85	0·5		0·2	0·3	Tremor, weakness and convulsions on eighth day. Died on ninth day. *Post mortem* examination showed no cause of death beyond poliomyelitis.
K26	4·07	0·5	0·4		0·1	No paralysis or symptoms.
K27	2·53	0·5	0·4		0·1	Complete paralysis ninth day. Killed when moribund on fifteenth day.

TABLE VIII.

Monkey.	Weight in Kilograms.	Virus in Cubic Centimetres.	Normal Serum in Cubic Centimetres.	Convalescent Serum in Cubic Centimetres.	Saline Solution in Cubic Centimetres.	Outcome.
K23	3·54	0·5			0·5	Prostrate on eighth day. Cord removed on tenth day.
K28	2·73	0·5		0·25	0·25	No paralysis or symptoms.
K29	2·82	0·5		0·3	0·2	Complete paralysis eighteenth day. Died twenty-second day.
K30	3·87	0·5		0·3	0·2	No paralysis or symptoms.
K31	3·25	0·5	0·5			No paralysis or symptoms.
K32	3·08	0·5	0·5			Complete paralysis ninth day. Cord removed on eleventh day.

TABLE IX.

Monkey.	Weight in Kilograms.	Virus in Cubic Centimetres.	Normal Serum in Cubic Centimetres.	Convalescent Serum in Cubic Centimetres.	Saline Solution in Cubic Centimetres.	Outcome.
K30	3·87	0·5			0·5	Complete paralysis nine to ten days. Cord removed on eleventh day.
K37		0·5		0·35	0·15	No paralysis or symptoms.
K39		0·5		0·35	0·15	Severe paralysis eleven to twelve days. Improved, but died on seventy-second day.
K40		0·5		0·35	0·15	No paralysis or symptoms.
K36		0·5	0·6			Doubtful very mild paralysis on twenty-first day. Prostrate on thirty-sixth day after typical attack. Died on fortieth day.
K38		0·5	0·6			Severe paralysis thirteenth day. Died on twenty-eighth day.
K41		0·5	0·6			No paralysis or symptoms.

TABLE X.

Monkey.	Weight in Kilograms.	Virus in Cubic Centimetres.	Normal Serum in Cubic Centimetres.	Saline Solution in Cubic Centimetres.	Outcome.
K37	3·7	0·5		0·5	Complete paralysis seven days. Cord removed ninth day.
K43	7·95	0·5	0·9		No symptoms or paralysis.
K44	6·15	0·5	0·9		Complete paralysis twenty-four days. Cord removed twenty-sixth day.

TABLE XI.

Monkey.	Weight in Kilograms.	Virus in Cubic Centimetres.	Old Convalescent Serum[11] in Cubic Centimetres.	Recent Convalescent Serum[44] in Cubic Centimetres.	Saline Solution in Cubic Centimetres.	Outcome.
K22	3·65	0·5			0·5	Complete paralysis eight to nine days. Cord removed on tenth day.
K37	3·60	0·5	0·5			No symptoms or paralysis.
K31	3·05	0·5		0·35	0·15	No symptoms or paralysis.

Owing to lack of monkeys, it was impossible to carry the investigation any further, but one further small experiment was performed to test the neutralizing power of an old batch of convalescent serum.

This Batch 21 was collected in April, 1928, from patients recently convalescent. Only three monkeys were available and of these one was used as a control, one was injected with a mixture of virus and old convalescent serum, Batch 21, and the third with a mixture of virus and a recent batch of convalescent serum (54) prepared in 1932. The results are shown in Table XI. It is evident that the five-year-old convalescent serum has retained definite virucidal power.

This experiment concluded the work on the neutralizing power of serum for poliomyelitis virus. A summary of the results obtained with convalescent serum is given in Table XII (excluding the single experiment with the old convalescent serum).

TABLE XII.

Volume of Convalescent Serum: 0·5 Cubic Centimetre of Virus Emulsion.	Number of Monkeys Inoculated.	Number showing no Symptoms or Paralysis.	Number showing Symptoms or Paralysis which Recovered.	Deaths.
0·1	2	0	1	1
0·2	2	0	0	2
0·25	2	2	0	0
0·3	2	1	0	1 (delayed)
0·35	4	3	0	1 (very late)

Thus, of four monkeys receiving 0·2 cubic centimetre or less convalescent serum, all were paralysed and three out of the four died. Of eight monkeys receiving amounts of convalescent serum between 0·25 cubic centimetre and 0·35 cubic centimetre to 0·5 cubic centimetre of virus emulsion, two were paralysed and died (in each case death being delayed, in one case to 22 days and the other 72 days), but the remaining six showed no symptoms at any stage. The neutralizing volume of convalescent serum for 0·5 cubic centimetre of virus emulsion is apparently somewhere in the region of 0·3 cubic centimetre.

The results obtained with pooled normal adult serum, which are given in Table XIII, are not so consistent.

The volume of normal serum necessary to neutralize 0·5 cubic centimetre of virus emulsion is evidently one rather greater than 0·6 cubic centimetre, since, of eleven monkeys receiving virus mixtures containing 0·5 cubic centimetre or more of serum, five showed no symptoms, two had definite though rather modified attacks of poliomyelitis, but recovered, and four died (death being delayed in three out of the four cases).

TABLE XIII.

Volume of Normal Serum: 0·5 Cubic Centimetre of Virus Emulsion.	Number of Monkeys Inoculated.	Number showing no Symptoms or Paralysis.	Number showing Symptoms or Paralysis which Recovered.	Deaths.
0·2	1	0	1	0
0·3	1	1	0	0
0·4	2	1	0	1
0·5	6	3	2 (1 delayed) (1 very mild)	1
0·6	3	1	0	2 (delayed)
0·9	2	1	0	1 (delayed)

Discussion.

In this method of investigating the neutralizing action of serum the occurrence of occasional survivals with small doses of serum is not significant. This may indicate either increased individual resistance, which is evidently subject to large variations, or possibly a diminished amount of virus in the mixture with serum. It must be recognized that we have no accurate quantitative measure of the dose of living virus present in the cord emulsion, and, in estimating the protective power of serum we are concerned only with the determination of that dose of serum which is invariably sufficient to neutralize all the virus present in a fixed volume of emulsion. The injection of such a mixture must never be followed by infection as indicated either by the production of symptoms or by a fatal result. If this end point be taken, individual variation of resistance is eliminated as a variable. The experiments recorded here, though few in number, indicate neutralization of a fixed volume of emulsion, which contains living virus varying between certain unknown limits, by rather more than 0·6 cubic centimetre of our normal serum and 0·3 cubic centimetre of convalescent serum.

Summary.

1. Pooled normal adult serum possesses a definite neutralizing action for poliomyelitis virus.

2. The neutralizing power appears to be between 30% and 40% that of convalescent serum.

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PART II.

THE RELATIONSHIP BETWEEN AUSTRALIAN AND AMERICAN STRAINS OF VIRUS.

Following the work of Burnet and Macnamara,(1) experiments were carried out on the relationship between American and Australian strains of poliomyelitis virus. Burnet and Macnamara found that monkeys which had been paralysed by the Melbourne strain LV (described in the first part of this paper, see Table I) were not immune to the Rockefeller "mixed virus" strain and concluded that there were definite immunological differences between the two strains of virus. We used a more viralent strain of local virus LVii (Table II) and obtained monkeys which had recovered from poliomyelitis following the inoculation of this strain. Some months later these monkeys were inoculated with a Rockefeller virus filtrate or emulsion. Temperatures were taken throughout, as it was thought that temperature curves might be valuable if the animals had an abortive attack without paralysis following the injection of the second strain of virus.

Details of Experiments.

The experiments were as follows:

Experiment I (Monkey K9).—On March 8, 1932, 0·5 cubic centimetre of Australian LV suspension was inoculated intracerebrally. Twelve days after the inoculation both legs were completely paralysed below the knee joint, but the arms and upper part of the body were unaffected. On April 13, 1932, the monkey was inoculated intracerebrally with 0·5 cubic centimetre of Australian LVii suspension, but showed no symptoms or paralysis as a result, although a control monkey was completely paralysed in seven days. Six months later (October 13, 1932) the monkey and a control monkey (K18) were injected with 0·5 cubic centimetre of a Rockefeller virus filtrate. The control became completely paralysed on the ninth day and died on the eleventh day after inoculation, but K9 showed no symptoms at any stage, and its temperature curve is that of a normal monkey (Figure II).

Experiment II.—(1) On December 8, 1932, three monkeys, K13, K17, K21, which had recovered from poliomyelitis developing after the inoculation of Australian LVii virus, and a control, K34, were inoculated intracerebrally with 0·5 cubic centimetre of Rockefeller virus filtrate. The previous histories of the monkeys were as follows:

K13.—The monkey was injected on July 13, 1932, with a mixture of Australian LVii virus and pooled normal serum, and as a result extensive paralysis developed. Gradual improvement took place, but the animal was still badly paralysed at the time of the second injection. A partial right facial paralysis was still present, there was wasting of the deltoid groups of muscles of the right shoulder and both legs were fixed in a flexed position at the knee joint and could not be straightened.

K17.—This monkey had been injected with normal adult serum and Australian LVii virus on September 7, 1932, and as a result had had a typical attack of poliomyelitis, although the paralysis was confined to one group of shoulder muscles.

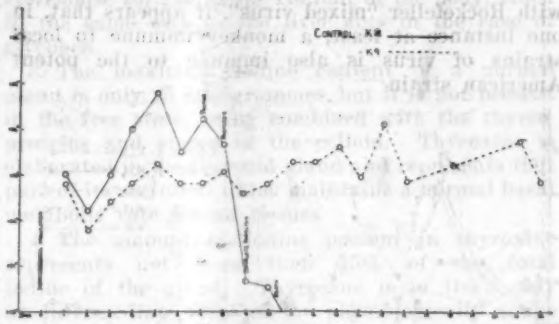

FIGURE II.
Temperature curves of monkeys K18 and K9 after inoculation with Rockefeller virus.

K21.—After inoculation with a mixture of convalescent serum and Australian LVii virus on October 26, 1932, the animal developed a tremor on the eleventh day and partial facial paralysis on the thirteenth day. No further paralysis developed, but the facial paralysis persisted and was present at the time of the injection of Rockefeller virus filtrate.

K34 was a normal monkey.

Most unfortunately, the Rockefeller virus filtrate used appeared to be completely inactive and produced no symptoms in the normal control K34 or in the three recovered monkeys. Figure III shows the temperature curves, which resemble those of normal monkeys.

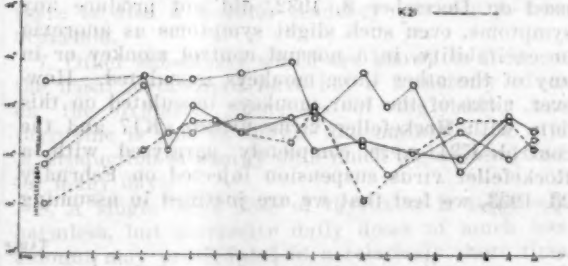

FIGURE III.
Temperature curves of monkeys K34, K13, K17 and K21 in experiment of December 8, 1932.

(2) On February 23, 1933, the same four monkeys and a new normal control, K42, were inoculated with a Rockefeller virus fresh cord suspension. The temperature curves of the five monkeys are shown in Figure IV. K42, the control, was completely paralysed by the seventh day, and K34 (the control for the experiment of December 8, 1932) by the tenth day after the injection. Of the three monkeys which had recovered after the injection of local virus, K13 and K21 showed no symptoms whatever, but K17 showed all the symptoms of poliomyelitis with a rise in temperature and tremor on the fourth day, a marked tremor and convulsions on the sixth day, and complete paralysis on the seventh day. The experiment is summarized in Table XIV.

Discussion.

From Experiment I it seems clear that the monkey K9 which had recovered from poliomyelitis after the inoculation of the moderately active strain of local virus LV was immune to the much more virulent local strain LVii. Further, since this monkey showed no symptoms when later injected with Rockefeller "mixed virus", it appears that, in one instance at least, a monkey immune to local strains of virus is also immune to the potent American strain.

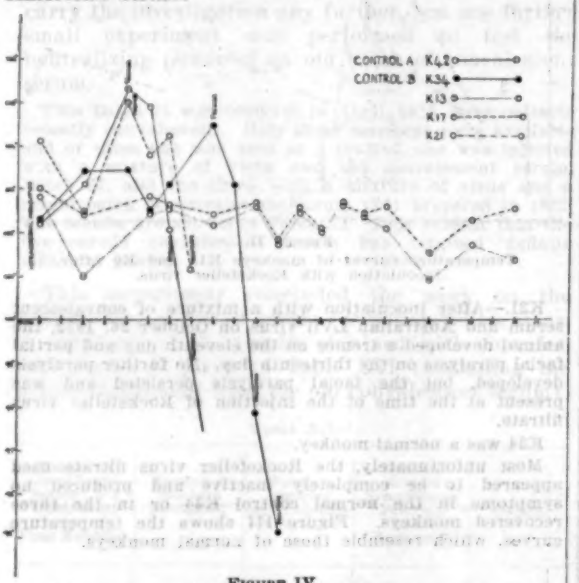

FIGURE IV.
Temperature curves of monkeys K34, K42, K13, K17 and K21 after inoculation with Rockefeller virus.

In Experiment II the Rockefeller virus filtrate used on December 8, 1932, did not produce any symptoms, even such slight symptoms as anorexia or excitability, in a normal control monkey or in any of the other three monkeys inoculated. However, since of the four monkeys inoculated on this date with Rockefeller virus filtrate, K17 and the control K34 were completely paralysed with a Rockefeller virus suspension injected on February 23, 1933, we feel that we are justified in assuming that the filtrate was inert and that its injection did not affect the immunity reactions of K13, K17 and K21. Flexner[2] reports in this connexion that:

> While one or more clinically ineffective doses of virus injected into the skin or subcutis increase the resistance or actively immunize monkeys, such non-infective injections made intracerebrally or intercisternally leave the inoculated animals essentially unchanged so far as response to more potent or larger doses of virus is concerned.

It appears, then, from Experiments I and II that of four monkeys paralysed by Australian strains, three (K9, K13 and K21) were immune to the Rockefeller "mixed virus" strain.

Burnet and Macnamara[1] found that three monkeys immune to a moderately active strain (LV) were not immune to Rockefeller virus and, conversely, that one monkey which had recovered with mild paralysis after the injection of Rockefeller virus was later completely paralysed by their local strain. They concluded that there was a definite immunological difference between the two strains of virus.

Stewart and Rhoads,[2] working with strains of different virulence, found that while intradermal inoculation protected against subsequent intracerebral inoculation with the same strain, it did not confer protection against a more virulent strain, suggesting the existence of different degrees of immunity according to the strength of the virus used.

Our few experiments seem to indicate that monkeys immune to a virulent strain possess a greater degree of immunity to the potent Rockefeller virus than do those immune to a weaker strain of local virus, and suggest that in further work on the relationship between Australian and American strains, the question of the virulence of the virus, as well as that of definite immunological difference, must be taken into consideration.

Acknowledgements.

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TABLE XIV.

Monkey.	Date of Inoculation with Australian LVii Emulsion.	Result.	Date of Inoculation with Rockefeller Virus Filtrate.	Result. (See Figure III.)	Date of Inoculation with Rockefeller Virus Emulsion.	Result. (See Figure IV.)
Controls for Rockefeller Virus. K42					February 23, 1933.	Tremor four days; complete paralysis six days. Cord removed seven days.
Controls for Rockefeller Virus. K34			December 8, 1932.	No symptoms or paralysis.	February 23, 1933.	Tremor eight days; complete paralysis ten days. Cord removed ten days.
K13	July 13, 1932.	Severe paralysis.	December 8, 1932.	No symptoms or paralysis.	February 23, 1933.	No symptoms or paralysis.
K17	September 7, 1932.	Mild paralysis.	December 8, 1932.	No symptoms or paralysis.	February 23, 1933.	Tremor four days; complete paralysis seven days. Cord removed seven days.
K21	October 25, 1932.	Mild paralysis.	December 8, 1932.	No symptoms or paralysis.	February 23, 1933.	No symptoms or paralysis.

Our thanks are also due to Dr. Dale, Chairman, and members of the Victorian Poliomyelitis Committee, and to Dr. F. G. Morgan and members of the technical staff of the Commonwealth Serum Laboratories.

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THE USE OF IODINE IN THYREOTOXICOSIS.

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AMONG those who come in contact with many patients suffering from toxic disorders of the thyreoid gland the term "exophthalmic goitre" is seldom heard, and it is not very often that patients are seen suffering from the classical signs of this malady. It is now customary to group disorders of the thyreoid gland under the general heading of thyreotoxicosis rather than that of toxic goitre, as in so many cases no enlargement of the thyreoid gland is apparent.

Very few practitioners seem to realize that there are many grades of toxic disorders of the thyreoid gland, for it appears to be an almost universal practice, once such a condition is even suspected, to order large doses of iodine and to give a favourable prognosis. Such methods are by no means confined to men practising in isolated country districts; in many instances learned senior physicians keep patients on iodine for lengthy periods, varying from three to sixteen months, despite the obvious fact that its immediate benefit has long since passed.

The experience of many clinicians has shown very definitely that when iodine is indicated its maximum effect is manifested within three weeks, and it is only in exceptional cases that any dose above 0·3 mil (five minims) of Lugol's solution three times a day is required. Yet within the last ten days I saw one patient who had been kept on 1·2 mils (twenty minims) three times a day for over three months, and another who had been taking ten to twelve drops with each meal for fourteen months.

For a partial understanding of the rôle played by iodine in thyreotoxic conditions it is necessary to review some of the salient points brought forward in the intensive investigations stimulated by the discovery of thyroxine in crystalline form by Kendall on December 25, 1914, and its synthetic preparation by Harrington in 1927. I use the words "partial understanding" advisedly, since there is no actual proof of the exact mode of action of iodine in toxic disorder of the thyreoid gland.

1. Appreciable amounts of iodine are present in all tissues of the body, and although these amounts are extraordinarily small, it seems that iodine is a necessity in the chemical reactions which are responsible for the physiological activity of normal tissues.

2. The thyreoid gland apparently acts as the storehouse for the iodine content of the body, much in the same way as the liver acts in the case of glycogen.

3. The maximum iodine content of a normal gland is only 25 milligrammes, but it is not present in the free state, being combined with the thyreoproteins and stored in the colloid. Thyroxine is elaborated in the thyreoid gland and represents that part of its secretion which maintains a normal basal metabolic rate for all tissues.

4. The amount of iodine present in thyroxine represents not more than 15% of the total iodine of the gland. Thyroxine is in itself only an intermediate form of the physiologically active compound required by the body tissues for their metabolic needs and is independent of the iodothyrin content of the gland, although it is firmly linked to the proteins of the thyreoid.

5. The daily requirement of thyroxine in the body is from 0·2 to 0·4 milligramme only, whilst the total amount functioning in the body is between eight and fifteen milligrammes, and this is responsible for approximately 40% of the total energy produced in the animal organism when at rest.

6. Any excess of thyroxine stimulates not only nitrogenous metabolism, but also that of carbohydrates and fat, that is, it increases the rate of oxidation of all metabolites, and in so doing it necessarily mobilizes the liver glycogen and renders the individual liable to a glycosuria and a possible ketosis. Owing to the drain on the alkali reserve associated with any form of increased metabolism, there is also a definite tendency towards a true acidæmia.

7. Other substances which are known to increase the basal metabolism have but a transitory effect on the oxidative processes of the body; but thyroxine is able to elevate and maintain the rate of production of energy on a much higher level and for many days.

8. A single large dose of thyroxine is relatively harmless, but successive daily doses of much less amount may prove fatal in a relatively short time while still smaller doses given over a prolonged period induce degenerative changes in all tissues concerned with the metabolic processes, that is, the liver, heart, kidneys and adrenal glands.

One or two other factors are of importance and are to be kept in mind when treating thyreotoxic conditions.

On a diet poor in calcium and potassium the thyreoid gland retains very litle iodine, so that it is necessary to insure a free supply of these substances or to supplement them in the diet, which should also be rich in vitamin *D* to assist in calcium metabolism. It is for this reason that

iodine should be given along with potassium, as has been found clinically in the use of Lugol's solution.

It should also be noted that the glycogen content of the liver is greatest on a diet rich in calcium. With any excessive thyreoid secretion, not only is there a diminution of the carbon dioxide combining power of the plasma, but the animal is rendered very susceptible to any lack of oxygen, this latter factor being an additional reason for being sure of a sufficient supply of glycogen, which combats a carbon dioxide deficiency. This also explains why clinical experience has taught us the vital necessity for avoiding any cyanosis when these patients are subjected to any form of inhalation anæsthesia, quite apart from the effect on a weakened heart of such a condition.

We are now in a position to see that there are several possible alterations in the iodine requirements of the body.

1. In conditions in which there has been any undue demand made on the thyreoid gland, such as at puberty, during pregnancy or even menstruation, at the climacteric, as a result of chronic general or local infectious or any exhausting illness, there may be increased activity of the gland, liberating unusually large amounts of thyroxine and other thyreoproteins into the system, that is, a true hyperthyreoidism. If this demand on the gland is long continued, the iodine supply may be exhausted and some dysthyreoidism result, thus paving the way for a thyreotoxic state, especially if the thyreoid gland is not normal.

2. In primary thyreotoxic conditions, for instance Graves's disease, there is a definite hyperthyreoidism to commence with, that is, an increase in both thyroxine and thyreoproteins. This continues to such an extent that the thyreoid gland cannot stand up to the demand made on its stored iodine, and thus colloid disappears from the gland. Iodine then ingested is eagerly seized upon and turned out incompletely metamorphozed in both thyroxine and thyreoproteins, and dysthyreoidism results. The tissues generally cannot secure iodine in the form in which it is best made use of, and an iodine deficiency occurs throughout the body. Hyperthyreoidism *plus* dysthyreoidism speeds up all metabolic processes, drives the heart, exhausts the somatic nerve cells, renders the sympathetic nervous system hypersensitive, mobilizes the liver glycogen and depletes the alkali reserve. If this is long continued, the blood pressure rises, the heart muscle becomes involved and chronic myocarditis ensues or auricular fibrillation develops.

When the iodine deficiency is made up in these patients the clinical results in most cases are very striking for the time being.

3. In secondary thyreotoxicosis alteration in the function of the thyreoid gland has been developing over a long period of time and results from either an adenomatous condition of the gland or a generalized fibrosis through the gland, consequent on either colloid goitre, a quiescent but old primary thyreotoxic condition, or possibly chronic sepsis. These conditions prevent stored colloid from becoming available to the tissues in suitable amounts and, from interference with the secretory activity of the gland, inhibit the metamorphosis of iodine, even though plenty be available in the diet. It is for this reason that in many of these cases iodine is not as beneficial as in the primary thyreotoxic condition, and must be given in small amounts so as to prevent an iodine flooding of the gland.

4. There is no doubt that large doses of iodine given to patients with adenomatous thyreoid glands may induce a thyreotoxic state, as excessive amounts of iodine are pushed on to a damaged gland which is incapable of dealing with them. As a result the gland swells, becomes firm, tender and even painful, and the so-called "iodine thyreoiditis" is produced, which acts as a precursor to dysthyreoidism and thyreotoxicosis.

In operating on such glands, dosed beyond their iodine capacity, the technical difficulties are greatly increased, since this chronic thyreoiditis affects not only the gland itself, rendering it extremely brittle, but causes fibrosis in the surrounding tissues and fixation of fascial planes.

Whenever iodine is given, the consistency of the thyreoid gland must be watched carefully, as once it becomes firm the dose should be reduced to a minimum or discontinued.

As iodine therapy plays such a big part in so many chronic diseases, a word of warning must be sounded to the physician to watch the thyreoid gland carefully. Many preparations of iodine are available to the public without any prescription, and patients often take large doses without any medical supervision. This, I feel sure, is one factor in the increasing number of secondary thyreotoxic conditions seen in recent years, and especially in women about the time of the menopause.

5. In some cases one sees patients who have been through a period of thyreotoxicosis and have arrived at a stage when they can carry on comfortably. This is a result of the adaptation of the body needs to a chronic dysthyreoidism and the attainment of a metabolic equilibrium sufficient for the needs of that particular individual for the time being. Even with a basal metabolic rate of + 80% it is possible to maintain a nitrogenous equilibrium, provided sufficient carbohydrates and fat are given. The caloric intake for such a purpose may be as high as 5,000 calories daily.

In very toxic states this equilibrium can be held only for a limited period, but with a lower basal metabolic rate of, say, + 15% to + 20%, it is possible to maintain a fairly stable balance over a period of years.

In such conditions it seems as if the toxicity of the thyreoid gland has burned out, but these cases always remind me of a quiescent volcano; gross damage has been done which can never be undone, and there is always the possibility of a further eruption, the violence of which it is impossible to

foretell. If the thyreoid gland of such a patient be flooded with iodine, a severe recrudescence of acute toxic symptoms may be induced.

For practical clinical purposes most cases of thyreotoxicosis can be readily classed as either primary or secondary. The classical example of the primary type is the acute Graves's disease. This is usually met with in patients between the ages of fifteen and twenty-five years, and only occasionally in later years.

It is in this condition that iodine has such a striking effect, but is never curative in itself. In these cases the basal metabolic rate is usually high and its significance must not be lost sight of. As already noted, increased metabolism connotes not only a general drain on the tissues and metabolites of the body, especially glycogen, but coincidently there is a great increase of the acids resulting from the oxidative processes with a consequent depletion of the reserve and a tendency to acidæmia.

This plays a very important part in the physical well-being of the individuals concerned, for if they are subjected to any unusual mental or bodily strain their condition is rendered worse; and if they are submitted without adequate preparation to anæsthetic or operation with their added disturbance of the hydrogen ion balance, a condition of true acidæmia may be induced, even to such a degree as to result in death.

In administering iodine we must bear in mind what we expect to achieve. A deficiency of this substance may occur in three directions, either singly or in combination, namely: (i) a general iodine deficiency in all tissues, (ii) a deficiency in the storage content of the gland itself, (iii) a deficiency in the iodine content of thyroxine, which is thus incompletely metamorphozed and leads to dysthyreoidism. After administering iodine the general well-being of the patient indicates that a general deficiency has been overcome. The gland becomes firmer when the colloid storage is brought about, and diminution of the basal metabolic rate and nervous symptoms is a sign that normal thyroxine is being elaborated; but this is the most difficult thing to overcome and regulate, as with a disordered gland hypersecretion *plus* dysfunction is apt to occur without apparent rhyme or reason.

It must therefore be obvious that once the maximum effect of administration of iodine has been achieved, the patient is fit for surgical treatment, provided the alkali reserve and glycogen storage have been remedied.

If iodine is persisted with, the damaged gland cannot cope with it and a chronic thyreoiditis is produced, leading to a further dysthyreoidism, which is very difficult to combat.

Treatment.

The first principle of treatment in thyreotoxic conditions is to insure complete rest, mental and physical, which can only be secured by strict confinement to bed. Plenty of good nourishing food must be taken, and this supplemented by additional glucose, alkalis, and possibly calcium and vitamin *D*. In the more severe cases give one drachm doses of an alkaline powder before each meal and at bed time, as well as a mixture with 1·2 grammes (twenty grains) of sodium bicarbonate after meals.

It is wise to add some drug having a sedative effect on the nervous system, such as sodium bromide, and if the required effect is not obtained with small doses of this drug or if patients show any idiosyncrasy to it, use can be made of "Barbenyl", "Luminal" *et cetera*.

Iodine is given in the form of Lugol's solution in doses of 0·12 to 0·6 cubic centimetre (two to ten minims) in milk with each meal, and if larger doses are necessary to start with, they are reduced to a minimum as the patient's condition improves.

By these means we induce a state in the patients which will bring them, as nearly as it can be done, into a state of equilibrium. If this happens to be something approaching the normal, it is permissible to regard it as sufficient and to allow that individual to carry on at this level as far as his walk in life will allow and for as long as fate permits.

If, however, it is found that the level of equilibrium is well above normal, as evidenced by a steady pulse rate of 100 or over and a basal metabolic rate of +20% or more, then further active treatment is demanded.

A few such cases will respond to X ray therapy, and I have seen some apparent cures, but again it seems that the attainment of a stable equilibrium compatible with a moderately active life, rather than an actual cure, is to be expected of such a form of treatment.

A skilfully performed operation by a man expert in this class of case is the quickest way we have at present of restoring the patient to as near normal as possible in such conditions.

Operation should never be performed until at least ten days' preparatory treatment has been carried out on the lines indicated, and not even then unless the patient has been brought into a fairly stable equilibrium. With the advent of basal narcosis these patients should never be allowed to have pre-operative worry or anxiety, at least in private practice, as it is impossible in a public hospital to conceal from them one's usual operating day.

As soon as the patient is returned to bed, 10% glucose solution and 1% sodium bicarbonate in saline solution is given *per rectum*, and when the patient can take it, plenty of fluid is given by mouth. I prefer paraldehyde as the basal narcotic in these cases as it induces a thirst, vomiting seldom occurs and the patients on return to bed are amenable to treatment and will take fluids by mouth with avidity long before they wake up.

With adequate pre-operative treatment I have very seldom found it necessary to give iodine *per rectum*, but would give small doses by mouth for about one week after operation. Sufficient morphine

is given to keep the patient quiet, as restlessness increases metabolism.

A policy of skilful neglect has been advocated for these cases, but I know of very few maladies which demand such patient, intensive watching and variation in treatment as a severe case of primary thyreotoxicosis.

When cardiac complications are present the responsibility is more than any one practitioner should shoulder, and he must insist on the closest cooperation with his colleague physician or surgeon, as the case may be.

I have dwelt at length on the subject of primary thyreotoxicosis as in most respects the treatment is very similar to that required in the secondary condition. The main difference in treating the latter is the administration of iodine, to which I have already alluded; moreover, the patient is liable to auricular fibrillation or myocardial degeneration, for which appropriate pre-operative treatment must be carried out in consultation with the physician.

In conclusion, I would advise against the use of belladonna, atropine or adrenaline in thyreotoxic conditions, as the sympathetic nervous system is already over-active, and these drugs are not only useless, but they may do great harm.

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O. A. A. Diethelm: "Pathological Conditions Associated with Disturbance of the Thyreoid Gland, with Special Reference to the Modern Views on Iodine Therapy", THE MEDICAL JOURNAL OF AUSTRALIA, May 26, 1928, page 634.

Reviews.

CARDIO-VASCULAR PAIN AND BIOCHEMISTRY.

DR. LAMBERT has had a very wide experience in clinical cardiology, including the periods when MacKenzie and Allbutt were the high priests of this branch of medicine, and his views are compounded of the best and most enduring of their doctrines, together with most of the recent developments and conclusions of the modern physiologists and clinicians. His monograph is a very complete summary of the more important contributions to the study of pain in heart disease by writers of the last and present century.[1] Adhering to the conviction that precordial pain in some instances can arise directly from the stimulation of sensory nerve endings in the aorta, though these occasions are in the minority, Lambert makes a plea for the substitution of the term cardiac pain by that of cardio-vascular pain. It is frequently impossible to distinguish which factor is chiefly responsible and both probably contribute a share, since aortic disease usually means stenosed coronary openings. Some of the statements used in support of his contentions are shrewd but quite hypothetical, while others are based on the unconfirmed results of one or two workers at variance with the majority, for instance, the view expounded by Russell that sclerosed arteries are particularly liable to spasm.

[1] "Cardiovascular Pain as a Biochemical Problem", by G. Lambert, B.A., M.D., B.C., with foreword by M. A. Cassidy, C.B., M.A., M.D., F.R.C.P.; 1933. London: H. K. Lewis and Company, Limited. Royal 8vo., pp. 86, with 23 illustrations. Price: 6s. net.

Gordon seems to hold the opinion that the unfavourable effect of infections such as influenza upon the heart is mostly exercised through the medium of their degenerative effects upon the arteries, and says little of the direct damage to the protoplasm of the muscle cells. At the same time he lays stress upon the fallacy of acquitting the heart as a cause of sudden death because no macroscopic disease is found at autopsy. In such cases the author emphasizes that a very complete histological examination is necessary. We know too little of the relationship between coronary mechanics and circulatory efficiency in the coronary arteries. Dr. Lambert makes much of the experimental work of Starling and Markwalder, who showed, physiologically at least, that anoxæmia causes not spasm, but dilatation of the coronaries, the corollary being that if coronary spasm is to be accepted as the cause of heart pain, some other blood condition than anoxæmia accounts for it. The biochemical problem is to determine the nature of this other factor.

Gordon considers that hepatic failure or derangement allows of a metabolic toxin or pressor substance to circulate in the coronaries and cause their contraction. Behind this, he considers there is an infective factor which has already damaged the coronary arteries. One of the valuable points made very clearly in this little book is the unity of all forms of precordial pain, including feelings of tightness and oppression, *angina minor* and classical *angina pectoris*. A clear outline of coronary physiology, an interesting survey of the evolution of our views on the cause of heart pain and a well illustrated description of the macroscopic neurology of the heart, all expressed simply and very well, make the contribution worth reading. One feels that the author has retained his scientific youth when he concludes with these words: "Biochemical research gives promise of adding to our knowledge and further compilation of statistics, based solely upon morbid anatomy, cannot yield the same results."

Notes on Books, Current Journals and New Appliances.

"CLINICAL SCIENCE INCORPORATING HEART."

TWENTY-FOUR years ago the journal *Heart* was established. Those who started the journal recognized that at that time cardiac problems were being studied with unusual intensity and that a journal specially designed to deal with the physiology and pathology of the cardio-vascular system was necessary. The original work in this field has diminished and the proprietors of the journal have decided to widen its scope in order to maintain its standard of publication. The title *Heart* has been altered to "Clinical Science Incorporating Heart". Though the proprietors are not to be congratulated on their choice of a title, they are to be commended for their enterprise. The new journal will appeal to a much wider circle of readers. The scope of the journal is shown by the articles in the first issue. H. P. Himsworth writes on the physiological action of insulin. Thomas Lewis and Werner Hess discuss pain from the skin and the mechanism of its production. E. J. Wayne makes clinical observations on two pure glucosides of digitalis, digoxin and *digitalinum verum*. G. W. Pickering records observations on the mechanism of headache produced by histamine. E. J. Wayne and L. B. Laplace contribute a paper on angina of effort, and F. H. Smirk writes on chloride and urea excretion as a measure of the functional activity of healthy and diseased kidneys.

The normal rate of publication will be one volume of approximately 400 pages a year. It will comprise four parts, to be published at irregular intervals. The subscription price will be thirty-seven shillings and sixpence, including postage. The publishers are Shaw and Sons, 6, 7, 8 and 9, Fetter Lane, London, E.C.4.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 23, 1933.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

HOSPITALS AND THE DEPRESSION.

In a recent issue of this journal reference was made to the public health and the present financial depression. The basis of the discussion was an article in the *Quarterly Bulletin of the Health Organisation.* One of the recommendations of the committee responsible for the report published by the League of Nations was that hospitals should be rationalized. As far as public health authorities are concerned, it was shown that there was need, not for curtailing activities in the sphere of preventive medicine, but for their extension. Health authorities are concerned primarily with the prevention of disease; hospitals with its treatment. Hospitals have, during the financial crisis, been faced with difficulties as great, if not greater, than those of health departments. Health departments are controlled by governments; and governments may, if they wish, shirk some of their responsibilities by cutting down expenditure and restricting the activities of their officers. We have shown that, by adopting such a course, governments are likely to increase the toll of disease; this will result in an increased demand by the public for hospital treatment, and, when hospitals cannot pay their way, the State will eventually have to foot the bill. Apart altogether from any possible increase in demand for hospital treatment consequent on a dereliction of departmental duty, more and more people are demanding hospital treatment on account of their reduced incomes.

The rationalization of hospital services is discussed in the report already referred to, under the following headings: (i) Standardization of hospital construction and equipment, (ii) rationalization of hospital management, (iii) establishment of cooperation between hospitals, (iv) rules governing the provision of hospital treatment and its duration, (v) cooperation between hospitals, public assistance authorities and health services. The question of standardization of hospital construction and equipment, cooperation between hospitals in regard to buying of equipment and provisions, in regard to management and questions of that kind need not be discussed in this place. They are matters purely for hospital boards; but it may be pointed out that cooperation in management should be considered. Attention should be paid to the suggestion that:

> It may be essential, especially in cities, to set up a central service to provide information regarding available beds in all the hospitals; this service will make a prompt and adequate allotment of patients to the various hospitals, specialised departments, homes, institutions for chronic diseases, convalescent homes, etc., so as to make the most rational use possible of the available beds and thus relieve the pressure on the hospitals.

It is natural and desirable that each hospital in a large centre should have its *amour propre*; at the same time hospitals are for the service of the public, and realization of a common purpose will lead to cooperation likely to bring fulfilment of that purpose.

In the several States of the Commonwealth there are differences in the organization and control of hospitals; in some places cooperation exists in certain of the directions indicated; it is thus possible to refer to this subject in only the most general terms. There is one adjunct to hospital practice and management, however, which must be mentioned. This is the establishment of a service of hospital almoners. Melbourne has seen the inauguration of such a scheme, but hospitals in other places have been slow to follow the Melbourne example. The functions of hospital almoners have

been described in these pages on previous occasions, and their usefulness to hospital, patient and medical attendant has been emphasized. The ground need not be covered again. At the same time, it must be stated emphatically that a hospital almoner service will be found most useful at the present time in furthering economy of time, of money and of effort in hospital practice. We would go further and state that when hospital almoners have taken up their work in any large centre, arrangements should be made, when necessary, for the exchange of information gathered by the almoners of different institutions. Until almoners are appointed to the staffs of all the large hospitals, the boards of management cannot be said to be doing their duty by the hospitals, the patients or the members of the medical staffs.

No mention has been made of cooperation between hospitals and such bodies as child welfare centres, baby clinics, the medical inspection branches of education departments and so on. Nor has reference been made to the determination of the type of patient who should be treated at hospitals. These are all of importance at the present time, but must be left for consideration to a future occasion.

Current Comment.

SCARLATINAL PURPURA.

PURPURIC manifestations may occur in a number of diseases from mechanical interference with the circulation or from an altered blood condition. In infectious diseases purpura has been noted in measles, variola, scarlet fever, typhus fever, epidemic cerebro-spinal meningitis and bubonic plague. It has also been seen in septic endocarditis, varicella and the paratyphoid fevers.

In the Lumleian Lectures of March, 1933, C. R. Box considers hæmorrhagic purpura as a rare sequela of scarlet fever.[1] But there is another type of hæmorrhagic scarlet fever resembling *purpura variolosa*, and the scarlatinal eruption is accompanied by severe purpuric manifestations. Box points out that in this the rash is hæmorrhagic and that profuse hæmorrhages occur from mucous membranes. In hæmorrhagic smallpox the prodromal scarlatiniform rash is hæmorrhagic and is accompanied by hæmorrhages from the mucous membranes with death even before the papular eruption appears. Some writers consider that similar cases of scarlet fever are actually variola.

[1] *The Lancet*, June 10, 1933.

Hæmorrhagic purpura in scarlet fever usually appears in the third week. The attack may have been mild and the purpura may be preceded by rhinitis, secondary tonsillitis or cervical adenitis, with albuminuria and slight pyrexia. Generally there is a sudden appearance of ecchymoses on the limbs, face and body, often near bony prominences and pressure points, possibly determined by slight trauma. The extravasations may become confluent and of a dusky purple or black hue. Sanguinous bullæ may form or necrosis and gangrene, probably from compression of the vessels. The unaffected skin may show waxy pallor out of all proportion to the blood lost. The purpura may extend to the mucous membranes, with ulceration and bleeding. Epistaxis is the most common; next, hæmaturia. Less often bleeding occurs from the stomach, bowels or elsewhere. Rarely the purpura is of Henoch's type, with colic. Cerebral involvement is rare. Hæmorrhagic softening of the temporal lobe has been noted as well as epileptiform fits.

It has been suggested that scarlatinal nephritis may be purpuric. Box states that this nephritis, without visible purpura, may be accompanied by abdominal colic, vomiting and constipation, as in Henoch's purpura. In post-scarlatinal purpura evidence of renal implication is often, but not always, present. Glomerular nephritis has been found after death from scarlatinal purpura, but scarlatinal hæmorrhagic nephritis usually runs its course without purpura. Necrosis may occur at the site of a large hæmorrhage, and gangrene of the nose, cheeks, fingers or toes has been noted, and rarely of one or both lower limbs. The association of purpura with gangrene is too frequent to be fortuitous. Gangrene of the limbs has been attributed to embolism with streptococcal endocarditis or associated with congenital syphilis. There may be no gross changes in the larger vessels. Box suggests that, possibly, abolition of the circulation by interstitial hæmorrhages determines the gangrene. Microscopic examination shows the tissues to be packed with red blood cells and polymorphonuclear cells, with compression of the small blood vessels. A hæmophiliac tendency has been suspected in some cases of determining the purpura, but any influence of hereditary or family tendency in precipitating this complication can, in Box's opinion, be excluded. Nor can he see any justification for the idea that the supervention of the hæmorrhagic condition can be attributed to the activation of a preexisting but latent hæmorrhagic tendency. He concludes that there must be some lesion engendered by the acute infection, but dependent on special circumstances for its manifestation.

Turning to the question of the pathology of the hæmorrhagic state, Box discusses capillary toxicosis and deficiency of blood platelets. He points out that there is ample evidence that the toxin of scarlet fever can inflict damage on the capillaries. In certain instances, for which there is no explanation, the hæmorrhagic nephritis of scarlet fever, so Box

believes, manifests itself as a purpura. Since to Box the proof of capillary damage appears to be strong, he looks for evidence of accompanying exhaustion of, or injury to the bone marrow. He deplores the inadequacy of blood examinations in this condition. There is evidence, however, that up to a certain point the bone marrow is stimulated to produce polymorphonuclear leucocytes, but beyond this point it fails rapidly. Box discusses the possible reasons for this and suggests that the exciting cause of the purpura is the scarlatinal toxin acting on the capillaries and small vessels, reinforced it may be by a similar toxic action on the bone marrow and having a special predilection for the megakaryocyte cells from which the platelets are formed. From this point Box goes on to consider the question of why the renal and purpuric manifestations are commonly delayed until the third week of the illness. The suggestion has been made that the kidney condition is the result of a hypersensitive state developing during the process of natural immunization following the scarlatinal infection. Box is logical in the view that what applies to the kidney might also apply to the purpuric lesions. Box's arguments need not be pursued further. He has opened up a subject of great interest, but one which, in the present state of our knowledge, cannot be explained. Comparatively little is known of the purpuric state, and it must not be forgotten that, the work of the Dicks and others notwithstanding, scarlet fever is by no means an open book. To go further than this at present is futile, but it should be emphasized that exhaustive clinical examination (particularly of the blood) and biochemical investigation may throw light on the subject.

TUMOURS OF THE SUPRARENAL GLAND.

RECENT reference has been made in this journal to the study of hypernephroma. From the clinician's point of view the term hypernephroma is useful, as it connotes a particular type of tumour, usually distinctive in appearance, yellow, often hæmorrhagic or necrotic in parts, and with some structural resemblance to the suprarenal cortex. The chief argument that has arisen over this group of neoplasms concerns their origin. The cordon-like arrangement of the cell columns originally suggested suprarenal "rests" as the source of these tumours, but this view has been strongly combated, as E. H. Derrick pointed out in his comprehensive survey in these pages over ten years ago. But there is another aspect of the subject of suprarenal tumours that is very interesting. The gland, as we know, consists of two parts distinct in origin and function. New growths are found that indubitably spring from one or other of these diverse structures, and these, in view of the modern interest in the ductless glands, are worthy of more than passing notice.

Charles F. Geschickter publishes an account of these primary suprarenal tumours, without including the so-called hypernephromata, which he regards as papillary renal carcinomata. He emphasizes the great difference existing between the two parts of the gland.[1] On the one hand we have the cortex, the "inter-renal organ", with its lipoidal cells, mesodermal in origin and concerned in some way with the functions of sex. On the other hand is the medulla, with its chromaffin cells, ectodermal in origin and allied to the sympathetic nervous system. The connexion between the malignant carcinoma of the cortex and precocious or pathological sexual changes is well known. Obesity, hirsutism and undue sexual development or alteration may occur, and it is interesting to recall that basophile adenomata of the pituitary gland cause similar symptoms and are sometimes associated with proliferation of the suprarenal cortex. Geschickter remarks that it seems probable that rapid proliferation is necessary in order to produce a definite alteration in the sex characters. This is interesting, for the usual experience with simple adenomata of the cortex, as he points out, is to find the growth accidentally at autopsy, without any hint of its presence during life. Does the overgrowth of cortex not supply enough putative hormone to alter the sex life or characters unless the rate of growth is rapid and the amount of new tissue considerable? It would seem so, yet thyreoid, pituitary, parathyreoid and pancreatic islet adenomata produce very definite symptoms related to over-production of the particular internal secretion. Apparently sexual change may occur with benign tumours, but is uncommon and very slight in degree.

The importance of these observations and the need for further work need only be mentioned. It is not merely a matter of curiosity on the part of the histologist; it concerns very nearly our growing, but still very defective, knowledge of the endocrines.

The medullary tumours are, of course, entirely different. A rare type of chromaffin carcinoma is found, and blood pressure changes may be observed during life, as might be expected. Apparently disturbance of the adrenaline output is at least one factor in producing vascular phenomena. Less uncommon is the malignant tumour arising from the sympathetic neural tissue of the suprarenal medulla, and this finds its counterpart in other similar tissues of the body, such as the great abdominal ganglia. These may range from the small-celled sympathoblastoma to the large-celled ganglioma. The commonest clinical type is the neuroblastoma occurring in young children. Patients suffering from this type of tumour have been demonstrated at various clinical meetings from time to time in the various States and reports published in these pages. Various clinical types of this growth have been described according as the metastases have been in the liver, the skull and other bones, or in the bone marrow. Geschickter considers this an unnecessary distinction, as the location of the secondary growths appears to depend upon the site of the primary only to a slight extent, but more particularly upon the age of the patient.

[1] *Archives of Pathology*, June, 1933.

Abstracts from Current Medical Literature.

MORBID ANATOMY.

The Renal Lesions in Hypervitaminosis D.

J. GOUGH, J. B. DUGUID AND D. R. DAVIES (*The British Journal of Experimental Pathology*, June, 1933) have investigated renal calcification. In hypervitaminosis *D* in the rat this condition usually appears as a granular deposit of calcareous matter scattered irregularly amongst the renal tubules, chiefly in the medulla. Sometimes it is accompanied by an extensive parenchymatous degeneration, described as nephrosis and somewhat resembling human chronic nephritis. The authors sought, by experiments with rats, to investigate the process of calcification and to discover what effect altering the reaction of the urine would have on its development. They varied the reaction of the urine by giving acid and alkaline orthophosphates. It was found that renal calcification generally went parallel with the amount of calcium excreted, and excretion of calcium was greatest when alkaline phosphate was given, and especially when calciferol was added. The urinary phosphorus excretion was greatest when the acid phosphate was given; calciferol lowered it, both when the acid and alkaline salts were given. The urinary excretions of calcium and phosphorus appeared to show a reciprocal relationship to one another. Though calcification and the parenchymatous degeneration, the so-called nephrosis, often coexisted, they were found to be largely independent of one another. Nephrosis could not, like calcification, be definitely correlated with any urinary disturbances noted by the authors. It seemed to be in a large measure dependent on the administration of acid phosphate with overdoses of vitamin *D*.

Congenital Cystic Disease of the Lungs.

D. H. COLLINS (*The Journal of Pathology and Bacteriology*, July, 1933) reports a case of congenital cystic disease of the lungs associated with a giant-cell hyperplasia of the lymph glands. The patient, a child of fifteen months, was admitted to hospital with bilateral enlargement of cervical and inguinal glands. Later on, death occurred from pneumonia following laryngeal diphtheria. The lungs were found at autopsy to have a diffuse honeycombed appearance, the condition being more pronounced in the upper lobes. The cysts were filled with purulent exudate, and their size varied from that of a pin-head to one centimetre across. No communication of the cysts with the bronchi was evident to the naked eye. The cysts were lined by flattened epithelium lying on a connective tissue base. No columnar epithelium was seen in any of the cysts, and nowhere was there evidence of communication between the cysts and the bronchi. In the mesenteric glands there was an epithelioid cell hyperplasia. Giant cells were present in great numbers; they resembled the giant cells of osteoblastoma. There was neither caseation nor tubercle formation. No tubercle bacilli were found. The ætiology of this condition is obscure; Grawitz believed it to be due to faulty development of lung lymphatics. The state of the glands remains unexplained.

Clinical Observations after Neoplasm Operations.

A. BITTDORF (*Münchener Medizinische Wochenschrift*, March 31, 1933) has seen secondary deposits occur many years after apparently successful operations for breast carcinomata and other neoplasms. Ten to fourteen years after the operation bone metastases may occur. The author records a case of a female, aged eighty years, with an enlarged, irregular liver, who, twenty-five years previously, had a melanosarcoma of the eye removed and who now had a large quantity of melanogen in the urine. He records another case of a woman who, eight years previously, had a left-sided breast carcinoma removed and then developed a right-sided pleurisy and a tumour shadow (walnut size) in the right lung hilus and also many small carcinomatous nodules in the skin. After six months' rest and diet, all these signs disappeared, the patient felt well and had gained weight. Several months later all the symptoms and signs recurred and a nodule removed from the mucosa of the mouth showed it to be a metastasis of a breast carcinoma.

Tissue Changes in Morbus Bang.

R. RÖSSLE (*Münchener Medizinische Wochenschrift*, January 6, 1933) writes that very little is known of the tissue changes in abortion fever occurring in man. He mentions a case of a butcher, aged thirty-seven years, who had sustained an injury to his left hand two months previously, followed by enlargement of the glands in the left axilla. Agglutination tests against *Brucella abortus* gave positive results. A gland was removed and sent for section, and three weeks later, owing to the persistence of fever, another attempt was made to excise a gland, but none was present, and therefore a piece of inflammatory material was removed for section. Microscopic examination of the gland showed that the gland was swollen, overfilled with cells and was surrounded by a thinned-out, tightly stretched capsule. The marginal and interfollicular sinuses were no longer seen, having been pushed away by the accumulation of cells. The lymphocytes were crowded together in the lymphoid nodules, and lymphoblasts could be seen in only a few spots. However, the most striking feature was the focal collection of cells very rich in protoplasm (probably mobilized reticular cells). At the periphery these cells were less sharply differentiated from the lymphoid tissue. These collections were rarely circular and usually exhibited many shapes, the characteristic cells being the large clear cells. Near the hilus, these cells became scattered in the tissue. The above findings of a regional adenitis are characteristic of that occurring in animals as well as in human beings. In a successful inoculation of an animal (guinea-pig is most useful) liver and splenic enlargement, as well as glandular enlargement, occurs. The bacillus of Bang can be recovered in the tuberculous-like foci in the liver and spleen. In a simple case there will be no difficulty in distinguishing by microscopic examination the lymph gland of abortion fever from that of lymphogranuloma or tuberculosis.

Lipoidgranulomatosis (Hand-Schüller-Christian Disease).

W. CEELEN (*Deutsche Medizinische Wochenschrift*, May 5, 1933) writes that Pick classifies the following diseases as primary disturbances of lipoid metabolism: Gaucher's disease, Niemann Pick's disease, Christian-Schüller's disease, xanthomata of varying and irregular localizataion to skin, mucous membrane and internal organs. The first two diseases belong to diseases of lipoid metabolism, but whether Christian-Schüller's disease belongs to this group is still not certain. The writer records a case of a child, aged two years, who complained of pain in the left knee and in the left leg. A little later soft areas were noticed in the head. Several teeth fell out and other teeth became loose. On palpation of the skull, a round, bony defect of five centimetres was felt, as well as numerous irregular, small, bony defects on the fifth and sixth ribs. A cherry-sized bony swelling was palpable on the right clavicle, and there was also fine crepitation of the clavicle. Both knee joints were swollen and painful on movement. X ray examination revealed numerous bony defects in the skull, thorax, pelvic bones and in the upper and lower extremities. *Post mortem* examination revealed focal disturbances in all the bones, and on section of the bones there were characteristic focal areas, sulphur yellow and hæmorrhagic in colour. The lipoid content of the adrenals was preponderant in the *zona glomerulosa*, the remaining cortex containing fat sparingly. Microscopic examination of the bones revealed a marked disturbance. The foci were partly myelogenous and partly periosteal, and consisted of a granulation tissue with predominant involvement of reticular cells, adventitial cells, histiocytes, lymphocytes, plasma and eosinophile cells. At the margin of the granulation tissue there were crowds of the well known fat-containing lipoid cells (xanthoma cells). By fat staining, these masses of fat were found to consist of lipoids and neutral fat and crystalline deposits. Whilst these lipoid cells were largely found in the bone, fatty

tissue and pleura, they were almost absent in the widespread focal and diffuse granulation tissue of the liver, lungs, lymph glands and spleen. In the last mentioned sites the proliferated reticular cells stored iron, but not fat. The author holds that his findings alone do not justify the assumption that the disease should be classed under the primary disturbances of lipoid metabolism. Whilst morbus Gaucher and Niemann Pick's disease are due to accumulations of lipoid, depending on a constitutional, more probably hereditary, foundation, this does not apply to Christian-Schüller's disease, since it may appear late in life and show a tendency to local improvement and healing. It is not known whether it is due to an infection or to chemical toxic influences. The question whether it is due to a primary lipoid deposit in the tissues or to a granuloma with secondary accumulation of lipoid requires further investigation and attention.

MORPHOLOGY.

The Influence of Pregnancies and Irradiated Ergosterol on Growth.

E. AGDUHR (*Upsala Läkareförenings Förhandlingar*, September, 1932) gives an account of the influence of pregnancies and of irradiated ergosterol on the growth of the body, as well as that of several organs, in two series of groups of white mice. One series was placed on a basal diet, while the other received in addition 0·2 milligramme of ergosterol (irradiated by ultra-violet light) per animal per day. In animals shut off from sexual intercourse these big quantities of ergosterol caused a retardation in the growth of the body, mainly a retardation in the development and the growth of the adipose tissue. In both series an increased number of the pregnancies caused a considerable augmentation of the body growth of the females, increased growth being more pronounced in the ergosterol series, where it was also proportional to the number of pregnancies undergone. Records were made of the effect on the liver, kidneys, lungs, heart and skeleton, as well as on the body as a whole. The vast majority of changes in growth are directed by several of the endocrine glands, which, as a consequence, show an increased or a decreased function, and this is indicated by an increase, retardation or a continuation of their normal growth. Increased sexual function in males appears to have a checking influence upon the growth of several organs of the body.

The Subdural Space and Its Linings.

T. LEARY AND E. A. EDWARDS (*Archives of Neurology and Psychiatry*, April, 1933), after reviewing the literature concerning the great differences in function and reactions of the pia-arachnoid and the *dura mater*, present evidence that the subdural space does not correspond to the other serous spaces, that the dura is not lined by mesothelium, but by fibroblastic tissue, and that the arachnoid is covered by cells which do not appear to be mesothelial, but which are probably of ectodermal origin, and to which the arachnoid apparently owes its impermeability. The readiness of the dura to produce adhesions if the arachnoid barrier is injured is thus accounted for. The dura, with its naked fibroblastic cells, is analogous to any exposed connective tissue surface, prepared to form granulations and adhesions unless restrained.

Surgical Anatomy of the Sensory Root of the Trigeminal Nerve.

L. DAVIS AND H. HAVEN (*Archives of Neurology and Psychiatry*, January, 1933) state that there are numerous anastomoses between the fibres of the sensory root of the trigeminal nerve near the hilus of the Gasserian ganglion. They find no topographical distribution of fibres in the sensory root which might be interpreted as an arrangement according to the type of sensation transmitted by them. There is a definite rotation of the sensory root in man and in animals between the Gasserian ganglion and the pons. Experimental section of the ophthalmic division of the ganglion and of the inferior portion of the sensory root immediately adjacent to the pons results in an identical microscopic picture of degeneration in the pons. Section in both instances is followed by a loss of corneal sensation and keratitis.

The Growth of the Epiphyses of the Long Bones.

C. G. PAYTON (*Journal of Anatomy*, April, 1933) attempts to solve the problem whether the epiphyses grow by additions to their diaphyseal surfaces, to their articular surfaces, or both. The present investigation offers evidence that the epiphyses grow at the side nearest the joint. He draws attention to a new observation on the appearance of the diaphyseal surfaces of the epiphyses which indicate absorption. This absorption is extensive, because the expected exceeds the actual increment by an average of 6·25 millimetres. Measurements of growth per week show that there is gradual diminution in the rate of growth in every epiphysis as one passes from the younger to the older epiphyses. The author also confirms the principle that the end of the shaft at which the epiphysis is last united, is the end at which growth takes place most quickly.

The Thoracic and Abdominal Distribution of the Vagus Nerves.

H. A. TEITELBAUM (*Anatomical Record*, February, 1933) gives an account of dissections of the vagus in the thorax and abdomen of new-born male infants. He stresses a nomenclature which takes account of the interchange between the left and right vagus nerves in the thorax. The cœliac plexus of the left side is described as consisting of an anterior and a posterior layer. The abdominal branches of the posterior vagus cord are classified on the basis of their origin as right anterior and terminal branches. On the basis of their relation to the cœliac ganglia these branches are classified as direct and indirect. The distribution of the direct branches of the posterior vagus cord to the aortic, phrenic, splenic, superior mesenteric, left renal, left internal spermatic and left suprarenal plexuses is described and illustrated on the basis of dissections.

The Cellular Content of Chyle.

R. D. BAKER (*The Anatomical Record*, February, 1933) gives an account of the lymphatics in the mesentery of the cat, and of an investigation into the cellular content of the prenodal and postnodal mesenteric lymphatics. It was found that the chyle almost always contained white cells before passing through the lymph node. Although the number of the cells per unit volume of chyle varied greatly, chyle which had passed through and around Peyer's patches contained on the average many more cells than chyle which had not. Furthermore, the number of cells in chyle was inversely proportional to the amount of fat present. This indicated that they had little to do with fat transportation in the lacteals. Chyle, after passing through a lymph node, contained, as a rule, more cells per unit volume than prenodal chyle. Wherever found, the cells of chyle were predominantly lymphocytes. The peripheral lymphoid tissue of the cat's small intestine appears to be analogous to a lymph node in respect to its capacity to release lymphocytes into lymphatic vessels.

Human Brachial Plexus United in a Single Cord.

E. SINGER (*The Anatomical Record*, March, 1933) describes an anomaly of the axillary artery associated with an irregularity of the brachial plexus, in which it appears as a single cord. Two similar cases had been previously reported. The indications are that the usual separation of the cords from each other is due to a normal development of the *arteria axillaris profunda* of the embryo, which persists in the mature individual as the *arteria axillaris*. The solid formation of the cord as observed in the case reported here is regarded by the author as a result of non-development or of retarded development of the fœtal *arteria axillaris profunda*. Under these conditions the existing *arteria axillaris* is a compensatory overdevelopment of the *arteria axillaris superficialis*. The article is accompanied by an illustration and a key of the condition described, and also a figure showing the constitution of the brachial plexus as revealed by tracing its fibres.

British Medical Association News.

MEETING OF THE FEDERAL COUNCIL.

A MEETING OF THE FEDERAL COUNCIL OF THE BRITISH MEDICAL ASSOCIATION in Australia was held at the British Medical Association House, 135, Macquarie Street, Sydney, on August 28, 1933. SIR HENRY NEWLAND occupied the chair.

Representatives.

The following representatives of the Branches were present:

New South Wales: Dr. J. Adam Dick, C.M.G., Dr. George Bell, O.B.E.
Queensland: Dr. D. G. Croll, C.B.E., Dr. E. S. Meyers.
South Australia: Sir Henry Newland, C.B.E., D.S.O., Dr. Bronte Smeaton.
Tasmania: Dr. Gregory Sprott, Dr. S. Gibson (as substitute for Dr. A. W. Shugg).
Victoria: Dr. F. L. Davies, Dr. J. Newman Morris.

Sir Henry Newland acted as proxy for Dr. F. A. Hadley and for Dr. D. D. Paton, of the Western Australian Branch.

Minutes.

The minutes of the final meeting of the Federal Committee of the British Medical Association in Australia, March 6 and 7, 1933, were read and signed as correct.

Financial Statements.

The financial statements of the Federal Committee for the six months ended June 30, 1933, prepared and audited by Messrs. Coates, Cunningham and Company, Chartered Accountants, together with the Australasian Medical Congress (British Medical Association) accumulated funds account, were presented and adopted.

Arrangements were made for the opening of bank accounts in the name of the Federal Council of the British Medical Association in Australia.

Appointment of President and Vice-President.

It was resolved, on the motion of Dr. J. Adam Dick, seconded by Dr. Bronte Smeaton, that Sir Henry Newland be appointed President and Dr. J. Newman Morris Vice-President of the Federal Council for the ensuing twelve months.

Appointment of Honorary Secretary and Honorary Treasurer.

No appointment was made to the position of Honorary Secretary.

On the motion of Dr. J. Newman Morris, seconded by Dr. Gregory Sprott, Dr. J. Adam Dick was appointed Honorary Treasurer for the ensuing twelve months.

Appointment of General Secretary.

The question of calling for applications for appointment to the position of General Secretary was discussed.

It was resolved:

> That a subcommittee be appointed to report on the matter at the next meeting of the Federal Council and that Dr. C. H. E. Lawes be appointed General Secretary for the ensuing twelve months.

It was also resolved:

> That the President and Dr. Bronte Smeaton be appointed a subcommittee to draw up the conditions of the appointment and to report at the next meeting of the Federal Council.

The Seal of the Federal Council.

The General Secretary reported that he had ascertained from the solicitors of the Federal Council that it was necessary that the Council should have a seal.

Dr. C. H. E. Lawes, Dr. J. Adam Dick, and Dr. George Bell were deputed to obtain an embossing seal and to make the necessary arrangements with regard to it.

Executive Committee.

The question of the appointment of an executive committee of the Federal Council in accordance with Article 13 was discussed. It was resolved that the appointing of an executive committee be deferred until the next meeting.

Incorporation of the Federal Council.

The certificate of incorporation of the Federal Council and the licence of the Governor-in-Council were produced and noted. The legal expenses in connexion with the incorporation were noted and the accounts were passed for payment.

The Broadcasting of Health Talks.

Letters on the subject of the broadcasting of health talks were read from the New South Wales and Victorian Branches. The opinion was expressed that some of the information given to the public over the air was unreliable, and it was thought that steps should be taken to remedy this defect.

It was resolved:

> That the health authorities be asked to restrict wireless lectures on health to legally qualified medical practitioners or other persons approved by the department in each State.

"Who's Who in Australia."

A letter was read from the Queensland Branch asking the Federal Council to express an opinion regarding the propriety of members of the Branches supplying information to the editor of "Who's Who in Australia".

Dr. J. Newman Morris could see no objection to the supplying of information. He pointed out that information about certain members would be obtained and would be published. It was much better that information published should be correct. He thought that they could trust the editor of the book not to publish information of a type that would be objectionable to the Association.

Dr. J. Adam Dick referred to the previous ruling of the Federal Committee on the matter. He quoted the resolution passed by the Federal Committee in 1915 as follows:

> That the Federal Committee, having given due consideration to the question of the propriety of responding to the invitation of the publishers of the "Medical Who's Who", expresses the opinion that it is undesirable for medical practitioners to permit personal notices of themselves to appear in such publications.

Dr. Dick thought that this decision of the Federal Committee should be reaffirmed and that the Australasian Medical Publishing Company, Limited, should be asked to proceed with the publication of a medical directory.

Sir Henry Newland, speaking a Director of the Australasian Medical Publishing Company, said that the company was not in a position to undertake the publication of such a work.

After considerable discussion and after perusal of the circular letter issued by the publishers of the book in question, a motion was submitted to the effect that the Federal Council saw no objection to members supplying the desired information. The motion was lost.

It was resolved, on the motion of Dr. E. S. Meyers, seconded by Dr. S. Gibson:

> That the matter be deferred for consideration by the Branches.

Members of the Association at Broken Hill.

At the last meeting of the Federal Committee the position of members of the Association at Broken Hill was discussed. These members had always belonged to the South Australian Branch; they wished to transfer their membership to the New South Wales Branch. It was pointed out at the meeting of the Federal Committee that it would be necessary to alter the Articles of Association of both Branches before the transfer could be effected. The General Secretary read correspondence from the New South Wales and South Australian Branches in which both Branches expressed their willingness to make the

necessary arrangements. The correspondence was received and it was noted that the matter lay between the two Branches in question.

Representatives of the Australian Branches on the Council of the Parent Body.

Reference was made to the valuable work done by Professor R. J. A. Berry and Sir Thomas Dunhill, as members of the Home Council representing the Australian Branches. It was resolved to send a letter to Sir Thomas Dunhill congratulating him on his knighthood. It was also decided that minutes of the Federal Council should be sent to Professor Berry and Sir Thomas Dunhill, in order that they might be kept informed of what was being done by the Branches in Australia.

"The Medical Journal of Australia" and the Branches.

A letter was read from the New South Wales Branch, forwarding a copy of a resolution as follows:

> That the Directors of the Australasian Medical Publishing Company, Limited, be requested to consult the Council of a Branch before matters of policy affecting that Branch are discussed in THE MEDICAL JOURNAL OF AUSTRALIA.

The correspondence was read and the matter was discussed.

The Editor of THE MEDICAL JOURNAL OF AUSTRALIA, on being invited to speak, explained to members of the Federal Council the liaison existing between the journal and the Branch Councils. He explained that the policy of the journal was necessarily the policy of the Federal Council. At the same time he insisted that neither the Federal Council nor the Branches should be regarded as being immune from honest criticism by any member who was prepared to sign his name to a letter embodying his views. He explained that the matter would be discussed by the Directors of the Australasian Medical Publishing Company, Limited.

It was resolved, on the motion of Dr. J. Newman Morris, seconded by Dr. George Bell:

> That the Directors of the Australasian Medical Publishing Company, Limited, be requested to take into consideration the question of the Editor consulting the Branch Councils, where possible, before matters affecting the policy of the Branch are discussed in THE MEDICAL JOURNAL OF AUSTRALIA.

Possible Influx of European Practitioners into Australia.

A letter was read from the Victorian Branch regarding the possible influx of European practitioners into Australia, owing to disturbed conditions on the continent of Europe. Dr. J. Newman Morris read a report on the matter by the Council of the Victorian Branch. It was explained that the question involved one of the reciprocity of registration. It was feared that practitioners from countries which had no reciprocal arrangements with the several Australian Medical Boards, might obtain registration in Great Britain and then transfer their registration to one of the Australian States. After discussion it was decided that the information supplied by Dr. Newman Morris should be sent to each Branch Council with a request that the Medical Board in each State be communicated with in the matter.

Confidential Information and Insurance Companies.

A letter was read from the Queensland Branch regarding the giving of confidential information to insurance companies. The Queensland Branch wished the Federal Council to reconsider the decision of the Federal Committee of August, 1926. It was explained that in August, 1926, the Victorian Branch forwarded to the Federal Committee certain resolutions, according to which it would not be unethical for a medical practitioner to give relevant information, provided that the proponent had signed a statement consenting to the giving of this information. The Federal Committee in 1926 disapproved of the resolutions of the Victorian Branch on the ground that by obtaining information in this way it might be possible for insurance companies to repudiate claims. The question was held to be important on account of the practice of some insurance companies of issuing policies without submitting the proponent to a medical examination.

Dr. E. S. Meyers advised that the Queensland Branch disagreed with the ruling of the Federal Committee.

It was resolved that the matter should be referred to the Branches and be brought up for discussion at the next meeting of the Federal Council.

Medical Officers' Relief Fund (Federal).

The Trustees of the Medical Officers' Relief Fund (Federal) forwarded a copy of the balance sheet as at June 30, 1933; of the revenue account for the twelve months ended June 30, 1933; and of the schedule of loans as at June 30, 1933. The statements were received.

A "Representative Body" in Australia.

At the last meeting of the Federal Committee a discussion took place on the desirability of forming in Australia a body analogous to the Representative Body of the Association in Great Britain, which might discuss medico-political matters affecting the Branches as a whole. The Federal Committee approved of the suggestion that the possibility of holding such meetings should be examined, and appointed Dr. D. G. Croll and Dr. E. S. Meyers as a subcommittee to formulate a scheme to be placed before the Federal Council.

After Sir Henry Newland had read the objects of the Federal Council, as set out in Clause 3 of the Memorandum of Association of the Federal Council, Dr. D. G. Croll said that he and Dr. Meyers had not drawn up a scheme. He went on, however, to explain the purport of the proposal, namely, that a body should be formed consisting of a representative of each local association in each State, and that the body so constituted should be called together at certain times, for example, at times when congresses were being held, to discuss matters of medical policy and to make recommendations to the Federal Council. It was thought that by this means members in outlying districts might be prevailed upon to take more interest in medical policy and that they would be given an opportunity of expressing their views. Dr. Croll thought that at a later date it might be possible to form a representative body, though it was not possible to do so at present.

After discussion it was resolved that further consideration should be deferred pending the submission of a report by the Queensland representatives.

Annual Meeting of the Association, 1935.

Reference was made to the annual meeting of the British Medical Association to be held in Melbourne in 1935. Dr. J. Newman Morris explained the steps that had been taken to date by the Victorian Branch. He said that the Victorian Branch wished all the Branches to take an active part in the organization of the meeting. To this end each Branch had been asked to nominate two members to act on the Executive Committee of the meeting. Dr. Newman Morris also said that the Victorian Branch wished to withdraw the question of a grant from the Federal Council, as it expected to be able to finance the meeting without assistance from the Federal Council.

Australasian Medical Congress (British Medical Association).

Correspondence from the Joint Honorary Secretaries of the Fourth Session of the Australasian Medical Congress (British Medical Association) was read. The appointment of Dr. D. H. E. Lines as president of the session was approved. Approval was also given for the date of the inaugural meeting of congress, January 15, 1934. The list of persons who had responded to the invitation of the Federal Council to be patrons of congress was noted. The vice-presidents of congress were appointed.

The advice of the Executive Committee of Congress was accepted, that the membership subscription should be three guineas for visiting and two guineas for non-visiting members.

Federal Health Services.

At the last meeting of the Federal Committee consideration was given to the public health administration in tropical Australia. At that meeting it was resolved that the Commonwealth Government should be urged to reestablish the Division of Tropical Hygiene or to institute an efficient substitute for it. A reply from the Minister of Health was read and members had before them a copy of *Health*, in which the activities of the Commonwealth Department of Health in tropical hygiene were set out. It was resolved that the Minister should be thanked for his reply and that the matter should receive further consideration by the Federal Council at its next meeting.

Australasian Pharmaceutical Formulary.

At the last meeting of the Federal Committee consideration was given to the suggestion of the Australasian Pharmaceutical Association for the publication of a formulary suitable for hospital and contract service. Dr. J. Newman Morris and Dr. F. L. Davies, at that meeting, were appointed a subcommittee to act in the matter and to report to the Federal Council. The subcommittee submitted its report and pointed out that the formulary was ready for publication. It suggested that the formulary should receive the approval of the Federal Council. The report of the subcommittee was adopted.

A Hospital Policy for the British Medical Association in Australia.

The question of the formulation of a hospital policy for the British Medical Association in Australia was before the last meeting of the Federal Committee. The matter had been brought up for consideration by the Queensland Branch. The Queensland Branch suggested that a subcommittee of the Federal Council should be formed to consider the matter. At the meeting of the Federal Committee the view was expressed that it might be possible to lay down certain general principles on which the Branches might act; the Federal Committee appointed Dr. D. G. Croll and Dr. E. S. Meyers a subcommittee to draw up a report on the matter for submission to the Federal Council.

Dr. Croll and Dr. Meyers reported that they had not been able to prepare a final report on the matter. They presented an *interim* report, which was received. The suggestion was made that arrangements should be made with the Executive Committee of Congress (Fourth Session) to set aside a time for discussion of hospital policy by members of the Branches.

It was resolved, on the motion of Dr. J. Newman Morris, seconded by Dr. George Bell:

> That, if the Executive Committee of Congress can find time, arrangements be made for selected representatives of the Branches to discuss hospital problems, and that the agenda paper and other details be left for the Hospital Policy Subcommittee with the approval of the Congress Executive.

An Insurance Contributory Scheme.

Consideration was given to a proposed insurance contributory scheme, put forward by the Colonial Mutual Life Insurance Company to the New South Wales, Queensland and Victorian Branches. Correspondence from these Branches was read and Dr. J. Newman Morris explained the scheme, which, he said, had been accepted by the Victorian Branch. He gave details of the premium, of the benefits, both in regard to hospital fees and fees for medical attendance, and discussed the advantages of the scheme.

It was resolved, on the motion of Dr. J. Newman Morris, seconded by Dr. S. Gibson:

> That the Federal Council approves of the hospital and medical insurance scheme of the Colonial Mutual Life Insurance Company as a definite scheme which recognizes medical services.

It was arranged that copies of the agreement of the insurance company with the Victorian Branch should be circulated by the Victorian Branch among the other Branches.

Repatriation Department.

A letter was read from the Chairman of the Repatriation Commission in reference to medical benefits for widows and orphans of soldiers whose death was due to war service, and widowed mothers of such deceased unmarried soldiers. It was pointed out that in 1924 the members of the medical profession had undertaken to treat these widows and orphans through the friendly societies at the rates and on the conditions obtaining in each district. It was held that such treatment was to be given to persons named in lists supplied at that time by the Repatriation Department. Since 1924 additional names had been added to the lists and the Federal Council held that this was not in accordance with the original agreement. The Chairman of the Repatriation Commission held opposite views. It was resolved:

> That a reply be sent to the Repatriation Commission reiterating the view of the Federal Council that no names should be added to the original lists as supplied in 1924.

A further communication from the Chairman of the Repatriation Commission had reference to the fees to be charged by local medical officers for the treatment of discharged soldiers. Certain alterations in the rates were approved, and it was noted that it would be necessary for the medical officer to send a separate account for each attendance. It was arranged that a copy of the letter from the Repatriation Commission, setting out the proposals, should be sent to the several Branches.

The Naval Medical Services.

The General Secretary read a report embodying the result of further inquiries made by him into the conditions of the Medical Services of the Royal Australian Navy. It was noted that the report of the committee appointed by the Prime Minister of Great Britain in June, 1931, "to investigate the causes of the shortage of officers and nurses in the medical and dental branches of the three defence services and to recommend by what means the situation can be remedied" was now available. It was resolved that the home Association should be asked to send to the Federal Council a copy of the report of the English committee, that when the report was received it should be summarized, and that the summary should be sent to the Navy Office in a letter to be approved by the President of the Federal Council.

Further reference was made to the correspondence which had taken place between the Federal Committee and the Director of Naval Medical Services regarding the treatment of injured dockyard workers. It was noted that the wishes of the Federal Committee had been met.

The Federal Health Council.

A letter was read from the Queensland Branch in reference to the Federal Health Council and suggesting that closer cooperation should be established between that body and the practising members of the medical profession. The matter was discussed and it was resolved, on the motion of Dr. E. S. Meyers, seconded by Dr. D. G. Croll:

> That the Federal Council recommends to the appropriate authorities that the Federal Council be allowed to nominate representatives, to be appointed by the Federal Government, on the Federal Health Council, as recommended by the Royal Commission on Health in 1925.

The Director-General of Army Medical Services.

A letter was received from the New South Wales Branch regarding the proposal that the Director of Army Medical Services should be a part-time officer. The New South Wales Branch expressed the opinion that such a step would be retrograde and not in the best interests of the Army Medical Services.

It was resolved:

> That in the opinion of the Federal Council the Director-General of Army Medical Services should be a whole-time officer of the Commonwealth.

Classification of Resolutions of the Federal Committee.

A notice of motion was received from the Queensland Branch suggesting that steps should be taken to issue in classified form the resolutions of the Federal Committee which affected more than one Branch of the British Medical Association in Australia, together with any other resolutions of the Federal Committee that, in the opinion of the Federal Council, should be added to the list. It was pointed out by the Queensland representatives that such a classification would be of immense value to those controlling the affairs of a Branch. It was resolved that steps should be taken to carry out the suggestion of the Queensland Branch.

Medical Officers to Sporting Bodies.

The Western Australian Branch forwarded a request for an opinion on whether the services of medical officers to sporting bodies should be paid or honorary. The correspondence was read. It was noted that all the other Branches had rules precluding members from accepting positions as honorary medical officers to sporting bodies.

It was resolved:

That a reply be sent to the Western Australian Branch to the effect that the Federal Council disapproves of the appointment of honorary medical officers to sporting clubs.

Thanks.

Votes of thanks were accorded to the Council of the New South Wales Branch for providing accommodation for the meeting and for hospitality extended to visiting members, and to the President, Sir Henry Newland, for presiding at the meeting.

ANNUAL MEETING.

THE ANNUAL MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Anatomy Theatre, University of Adelaide, on June 28, 1933, DR. F. ST. JOHN POOLE, the President, in the chair.

Annual Report of Council.

The annual report of the Council was received and adopted. The report is as follows.

Election.

At the annual meeting held last June the following were elected:

President: Dr. F. St. John Poole.
Vice-President: Dr. E. Britten Jones.
Honorary Medical Secretary: Dr. Alan H. Lendon.
Honorary Treasurer: Dr. W. A. Verco.
Members of Council: Dr. H. Halloran, Dr. L. C. E. Lindon, Dr. Allan D. Lamphee, Dr. A. F. Stokes.
Federal Committee: Sir Henry Newland, Dr. Bronte Smeaton.

At the first meeting of the Council, held on July 7, 1932, the following subcommittees were appointed:

Scientific: The President, Sir Henry Newland, Dr. E. Britten Jones, Dr. W. A. Verco, Dr. L. C. E. Lindon (Honorary Secretary, convener).
Contract Practice: The President, Dr. H. Powell, Dr. H. Halloran, Dr. A. F. Stokes (Lay Secretary, convener).
Ethical: The Council.
Library: The President, Sir Henry Newland, Dr. P. Gorrie, Dr. Allan D. Lamphee (Honorary Secretary, convener).
Parliamentary Bills: The President, Dr. E. Britten Jones, Dr. A. V. Benson (Lay Secretary, convener).
Revision of Rules: The President, Sir Henry Newland, Dr. Bronte Smeaton (Lay Secretary, convener).

Meetings.

The Council.—The Council met on twelve occasions, the attendance being:

Dr. A. V. Benson	10	Sir Henry Newland (on leave)	2
Dr. P. Gorrie	2	Dr. F. St. John Poole	12
Dr. H. Halloran	11	Dr. H. Powell	7
Dr. E. Britten Jones	10	Dr. Bronte Smeaton	11
Dr. Alan H. Lendon	12	Dr. A. F. Stokes	11
Dr. L. C. E. Lindon	11	Dr. W. A. Verco	2
Dr. Allan D. Lamphee	10		

Scientific Subcommittee.—The Scientific Subcommittee met once, the attendance being:

Dr. E. Britten Jones. Dr. F. St. John Poole.
Dr. Alan H. Lendon. Dr. W. A. Verco.
Dr. L. C. E. Lindon.

Contract Practice Subcommittee.—The Contract Practice Subcommittee met on eight occasions, the attendance being:

Dr. H. Halloran	8	Dr. F. St. John Poole	8
Dr. H. Powell	5	Dr. A. F. Stokes	8

Library Subcommittee.—The Library Subcommittee met once, the attendance being:

Dr. Allan D. Lamphee. Dr. Alan H. Lendon.
Dr. F. St. John Poole.

Monthly General.—Nine meetings were held during the year, the attendances throughout being satisfactory. The September meeting was held at Victor Harbour. A golf competition was arranged for the morning and afternoon, which added to the enjoyment of those who attended, and the meeting was successful in every way. The Council appreciates the action of the Victor Harbour Golf Club in making their links available to members; also the assistance given by Dr. F. J. Douglas and Dr. G. S. Shipway.

The following programme was carried out:

1932—

July: Paper by Dr. Ian Hamilton on "Some Aspects of Gall-Stone Disease". (Discussion opened by Dr. P. S. Messent.)
August: Paper by Dr. Gilbert Brown on "Premedication with Barbiturates". (Discussion opened by Dr. Allan D. Lamphee.)
September: Paper by Dr. W. Ray on "The Investigation of Patients with Dyspepsia". (Discussion opened by Dr. F. Ray Hone.)
October: Paper by Dr. F. H. Beare on "The Pituitary Syndromes". (Discussion opened by Dr. Guy Lendon.)
November: Paper by Dr. R. F. Matters on "Benign and Malignant Conditions of the Cervix". (Discussion opened by Dr. Roland Beard.)

1933—

February: Paper by Dr. Alec L. Dawkins on "The Modern Treatment of Varicose Veins and Ulcers". (Discussion opened by Dr. A. Britten Jones.)
March: Paper by Dr. D. R. W. Cowan on "Pulmonary Tuberculosis, with Special Reference to Earlier Aspects". (Discussion opened by Dr. Frank S. Hone.)
April: Description by Sir Henry Newland of the British Medical Association Centenary Meeting held in London last year.
May: Listerian Oration by Dr. F. A. Maguire, of Sydney, on "Gynæcological Problems Considered in the Light of Listerism".

During October Dr. Reg. S. Ellery, of Melbourne, also addressed members on "Neurosyphilis". The meeting was arranged by the late Dr. M. H. Downey at the Parkside Mental Hospital.

Listerian Oration.

The oration this year was delivered by Dr. F. A. Maguire, of Sydney, on May 25, his subject being "Gynæcological Problems in the Light of Listerism". An excellent gathering of members attended, and the Council desires officially to thank Dr. Maguire.

Membership.

The membership of the Branch is 377. The number of new members elected was 10, the balance representing the difference between transfers "in and out", after deducting deaths.

It is with deep regret that we record the deaths of Dr. T. Gibson, Dr. Hampden Carr, Dr. J. R. Muirhead, Dr. A. F. Lynch, Dr. M. H. Downey, Dr. N. R. Mathews, Dr. H. F. Shorney, and Dr. R. Douglas Brummitt. The Council also regrets the death of Dr. R. Humphrey Marten, a member of the Association for many years and President during the year 1898-1899.

The Council has elected Dr. L. B. Bull as a complimentary member of the Association.

Representation on Boards.

Dental Board.—Dr. P. S. Messent was appointed to represent the Branch on the Dental Board.

Nurses' Board.—Dr. S. R. Burston has been appointed to represent the Branch on the Nurses' Registration Board for a further term of three years. During his absence in England, Dr. H. H. E. Russell will act on the Board as representative of the Branch.

Saint John's Ambulance Association.

The Saint John's Ambulance Association asked the Council to appoint a direct representative of the Branch on their Committee, and Dr. A. V. Benson was appointed.

Federal Committee.

Dr. Bronte Smeaton represented the Branch at the meeting in Melbourne on March 6, 1933, Sir Henry Newland being absent on leave. This was the last meeting of the Committee, which has now been replaced by the Federal Council.

The passing of the Federal Committee is noteworthy. It has been in existence for twenty years, and recalls the debt of gratitude owing to those whose vision and devotion did so much to weld the medical profession of the Commonwealth into a united body. The Committee has been the connecting link between the parent Association and the Branches, and has by its achievements done much, within the scope of its powers, to promote medical and allied sciences.

Federal Council.

The Federal Committee, having decided that the Federal Council should be incorporated and that certain amendments to the proposed Memorandum and Articles of Association required by the Minister for Justice should be made, completed copies of the Memorandum and Articles of Association were signed by the members of the Committee, and the signatures duly witnessed at the meeting held on March 6 last, which was the last meeting of the Federal Committee. The Memorandum and Articles of Association have now been signed by the Governor of New South Wales. The next time that representatives of the Branches meet, they will meet as the Federal Council, which will be able to more effectively carry out the objects of the British Medical Association on behalf of the various Branches.

Work of the Sections.

Eye, Ear, Nose and Throat Section.—The Eye, Ear, Nose and Throat Section has held eight meetings during the year, the attendance being satisfactory.

Clinical Medicine.—Six meetings of the Section of Clinical Medicine were held during the year and were well attended.

Anæsthetics.—The Section of Anæsthetics met three times during the year.

History of Medicine.—The section of the History of Medicine was inaugurated in November, 1931. The membership is 26. Four meetings have been held during the year.

Surgery.—At a meeting of the Council held on August 4 last, permission was given for the formation of a Section of Surgery. The inaugural meeting was held on November 9, 1932.

Lodge Matters.

The Council has been in close touch with lodge matters during the year. The Contract Practice Committee met eight times, but the various developments from time to time necessitated a good deal of attention. There has been no alteration in the rate in the metropolitan area and, as far as the Council is aware, the country members have maintained the rate which existed last year. Although improvements might be made in regard to certain conditions obtaining at present, which are largely the result of the existing financial stringency, it is expected that means will be taken to overcome the problem of the unfinancial members, unsatisfactory lists, late payments *et cetera*. It is very desirable that lodge surgeons will adhere to the conditions laid down in the New Model Agreement, as any departure from the agreement will undoubtedly lead to greater difficulties in any future negotiations.

Adelaide Permanent Post-Graduate Committee.

The annual refresher course was held from May 22 to 28 last. The "E. C. Stirling Lectures" were delivered during the course by Dr. H. Douglas Stephens. The interest in the course was well maintained, a representative number taking advantage of the lectures.

Library.

Country members have been notified that arrangements have been completed with the university which will enable them to borrow books from the library, but only a few have taken advantage of the privilege. In bringing this again under the notice of members in the country districts the Council hopes that many more will avail themselves of the opportunity of borrowing books under the conditions granted.

The Library Committee have recently recommended additional books to the library. During the year the parent Association forwarded to the Branch a gift of ten books of the "Hastings Collection" from the library at Worcester, founded by Sir Charles Hastings. These books are of great historical interest.

British Medical Association.

An outstanding event during the year has been the centenary of the Association, which was formed in 1832 by Sir Charles Hastings. The meetings held in July last were attended by delegates from many parts of the world. The Branch was represented by Sir Henry Newland as representative, and Dr. S. L. Dawkins and Dr. J. Stanley Verco as delegates. The appointment of Sir Henry Newland as a Vice-President of the British Medical Association and Vice-President of the Section of Surgery, also Dr. Gilbert Brown as Vice-President of the Section of Anæsthetics, was a great honour to these members and also to the Branch.

It is of interest to note that in connexion with the centenary meeting a conference of representatives of overseas Branches and Divisions was included in the programme as an experiment, and advice has now been received that, in view of the success of the conference, it has been decided to hold another such conference in connexion with the annual meeting, 1934.

It is noted with great pleasure that in all probability the annual (1935) meeting of the British Medical Association will be held in Melbourne in September of that year, and the event will be one of great importance to the members of the British Medical Association in Australia.

Australasian Medical Congress, Fourth Session.

It has been decided to hold the Fourth Session of the Australasian Medical Congress (British Medical Association) in Hobart, January 15 to 20, 1934, and Dr. Alan H. Lendon has been appointed the Honorary Local Secretary.

"The Medical Journal of Australia."

The journal has maintained its high standard of previous years. The wish of the Editor and Directors is to make it as useful as possible to members of the Branches, and THE MEDICAL JOURNAL OF AUSTRALIA, is therefore the personal concern of every member.

South Australian Joint Committee for Combating Infantile Paralysis.

The Joint Committee of the Branch and Red Cross Society formed in 1930 has carried on its work successfully. The report for the year 1931-1932 has been sent to members, and this gives full particulars of the activities of the committee.

Tuberculosis.

On November 5, 1931, the Council appointed a committee to report on the Consumptive Home at Northfield, and this was submitted to the Council on February 4, 1932. In view of the importance of the question of tuberculosis in its relation to the State, the Council felt that wider scope should be given to the Committee, who were delegated to inquire into tuberculosis with a view to approaching the Government regarding better control in this State.

The Committee made exhaustive inquiries, meeting on fourteen occasions, and have submitted a full report, with recommendations, for the consideration of the Council. As a direct result of the work of the Committee, the Commonwealth Employment Council has agreed to make a grant of £3,000, and the State Government £1,500, for the erection of a tubercular clinic in the Adelaide Hospital grounds. Plans for the building have been approved, and it is expected that the work will be started shortly.

Farmers' Relief Acts.

Efforts have been made to improve the conditions that existed in regard to doctors' accounts under the *Farmers' Relief Acts*. At the request of the Council, the President interviewed the Chairman of the Debt Adjustment Commission and discussed with him the procedure in respect to payment for medical services under the acts. As a result of the representations made by the Council, improvement has taken place, and at present a more equitable distribution has been adopted, giving more satisfactory results to doctors who have attended patients financed under the Relief Acts.

Resignation of Dr. W. A. Verco as Honorary Treasurer.

At the February meeting of the Council the resignation of Dr. W. A. Verco as Honorary Treasurer was received. The Council regretted that he felt compelled to take this step owing to ill health, but asked him if possible to carry on until the annual meeting in June, to which he consented. Dr. Verco was appointed in 1914 and has held office continuously since that time. The Branch has been fortunate in having his wise counsel in its financial affairs during these years. At a meeting of the Council held on June 1, reference was made to the work of Dr. Verco and the sincere appreciation of the Branch of his services was placed on the records.

General Remarks.

The year has been a busy one, close attention and earnest consideration being given to various matters affecting the interests of the medical profession. The extension of the activities of the Branch is a pleasing feature of the year's work. Your Council asks for the earnest cooperation of all members in the work of the Association, and hopes that those who have not taken any interest in the welfare of the Branch will, by their attendance at the various meetings, give greater support to the advancement of the British Medical Association.

(*Signed*) F. ST. JOHN POOLE,
President.

Financial Statements.

The financial statements, as submitted by the Honorary Treasurer, Dr. W. A. Verco, were received and adopted. The statements are published herewith.

Income and Expenditure for Year ended December 31, 1932.

	£ s. d.	£ s. d.		£ s. d.
To Amount due British Medical Association, London, on Paid Subscriptions	407 10 6		By City Subscriptions	843 15 0
„ Amount due British Medical Association, London, on Unpaid Subscriptions	58 13 0		„ Country Subscriptions	475 17 6
		466 3 6	„ Accrued Subscriptions	189 3 0
„ THE MEDICAL JOURNAL OF AUSTRALIA		420 0 0	„ Interest	17 18 9
„ Library Account		56 0 0	„ Sundries	0 3 1
„ Telephone	27 13 10			
„ Exchange	2 9 6			
„ Rent	52 0 0			
„ Postages and Telegrams	29 10 1			
„ General Expenses	44 5 11			
„ Stationery and Printing	32 3 0			
		188 2 4		
„ Lister Medal		0 10 0		
„ Federal Committee Expenses		22 5 8		
„ Salary		350 0 0		
„ Depreciation		12 1 6		
„ Balance		11 14 0		
		£1,526 17 4		£1,526 17 4

Library Fund Account, December 31, 1932.

	£ s. d.		£ s. d.
To Adelaide University	50 0 0	By Balance brought down from December 31, 1931	517 17 7
„ Depreciation	12 4 8	„ Interest from Savings Bank	4 6 5
„ Balance	516 9 4	„ Country Subscriptions	0 10 0
		„ Transfer from Income and Expenditure Account	56 0 0
	£578 14 0		£578 14 0

General Fund Account.

	£ s. d.		£ s. d.
To Telephone Account for 1931	8 13 2	By Balance brought down, December 31, 1931	2,812 2 1
„ Federal Expenses for 1930 and 1931	13 1 0	„ Shares in British Medical Hall Company, Limited, presented by members	30 0 0
„ Balance	2,832 2 3	„ Income and Expenditure Account	11 14 4
	£2,853 16 5		£2,853 16 5

Balance Sheet as at December 31, 1932.

Liabilities.	£ s. d.	£ s. d.	Assets.	£ s. d.	£ s. d.
To Subscriptions paid in advance		8 2 0	By Plant and Fittings	120 15 4	
„ Amount due British Medical Association, London, on Paid Subscriptions	1,395 6 0		*Less* Depreciation	12 1 6	
„ Amount due British Medical Association, London, on Unpaid Subscriptions	58 13 0				108 13 10
		1,453 19 0	„ Investments—		
„ The Medical Journal of Australia		113 2 6	Commonwealth Loan	403 12 0	
„ Library Fund		516 9 4	British Medical Hall Company, Limited	1,950 0 0	
„ British Medical Association, London, Savings Bank Interest		55 18 1			2,353 12 0
„ Sundry Creditors		52 8 6	„ Library Fund—		
„ General Fund		2,832 2 3	Delineascope	50 16 7	
			Projector and Kodascope	71 10 0	
				122 6 7	
			Less Depreciation	12 4 8	
					110 1 11
			„ Savings Bank Library Fund		128 6 5
			„ Lister Medals and Dies		19 13 4
			„ British Medical Hall Company, Limited, Loan		450 0 0
			„ Subscriptions owing		255 16 0
			„ Stocks—		
			Hospital Forms	0 15 7	
			Medical Certificate Books	4 5 9	
					5 1 4
			„ Sundry Debtors		103 5 0
			„ Cash—		
			State Savings Bank	1,000 0 0	
			Commonwealth Savings Bank	436 18 3	
			National Bank of Australasia, Limited	54 10 0	
			In hand	6 3 7	
					1,497 11 10
		£5,032 1 8			£5,032 1 8

Audited and found correct.

C. W. L. MUECKE,
Chartered Accountant (Aust.),
Auditor.

Adelaide,
May 18, 1933.

W. A. VERCO, Honorary Treasurer.
WALTER C. DOBBIE, Lay Secretary.

Induction of President.

Dr. F. St. J. Poole introduced the President, Dr. E. Britten Jones, and wished him a successful term of office.

Dr. E. Britten Jones thanked the members for having elected him to the presidential chair.

Election of Office-Bearers.

Office-bearers for the ensuing twelve months were declared elected as follows:

Vice-President: Dr. L. C. E. Lindon.
Honorary Medical Secretary: Dr. Alan H. Lendon.
Honorary Treasurer: Dr. F. St. John Poole.
Delegates to the Federal Council: Sir Henry Newland, Dr. Bronte Smeaton.
Members of Council: Dr. G. H. Burnell, Dr. A. R. Clayton, Dr. D. R. W. Cowan.

President's Address.

Dr. F. St. John Poole then delivered his presidential address (see page 395).

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Gammie, Charlotte Isabella, M.B., B.S., 1928 (Univ. Sydney), Women's Hospital, Crown Street, Surry Hills.
Gardiner, Thomas Joseph, M.B., B.S., 1929 (Univ. Melbourne), Young.
Nelson, Willa, M.B., B.S., 1930 (Univ. Sydney), Blayney.
Saxby, Noel Hunter West, M.B., B.S., 1930 (Univ. Sydney), Royal Hospital for Women, Paddington.

The undermentioned have been elected members of the Tasmanian Branch of the British Medical Association:

Robertson, J. R., Deloraine, Tasmania.
Jeffrey, Eric, 4, Douglas Avenue, Sandy Bay, Tasmania.

Post-Graduate Work.

ANNUAL REFRESHER COURSE IN MELBOURNE.

THE Melbourne Permanent Post-Graduate Committee has announced the details of the annual refresher course to be held in Melbourne from November 13 to 25, 1933. The fee for the course is three guineas.

Arrangements have been made with the Committees of Management of the Melbourne and Alfred Hospitals whereby a limited amount of accommodation will be available at these hospitals for those attending the course, at an additional fee of £3 3s. per week to cover board and lodging.

The time-table is as follows.

Monday, November 13, 1933.

9 to 9.30 a.m.—Melbourne Hospital: Registration at Post-Graduate Office, Melbourne Hospital.
9.30 to 11 a.m.—Melbourne Hospital: Dr. J. Ivan Connor, "The Treatment of Superficial Staphylococcal Infections".
11.15 a.m. to 12.45 p.m.—Melbourne Hospital: Dr. G. C. Scantlebury, throat, nose and ear cases.
2.15 to 3.30 p.m.—Melbourne Hospital: Dr. H. Hume Turnbull, "Lesser Forms of Cardiac Infarction".
3.30 to 5 p.m.—Melbourne Hospital: Dr. B. T. Zwar, "Management and Treatment of Prostatic Enlargement".
8.30 p.m.—B.M.A. Hall: Professor F. Wood Jones, Lecture I.

Tuesday, November 14, 1933.

9.30 to 11 a.m.—Saint Vincent's Hospital: Dr. W. J. Newing, "Artificial Pneumothorax".
11.15 a.m. to 12.45 p.m.—Saint Vincent's Hospital: Dr. Leo Doyle, "Hints for Local Anæsthesia".
2.15 to 3.45 p.m.—Eye and Ear Hospital: Oto-rhino-laryngology.
4 to 5.30 p.m.—Eye and Ear Hospital: Ophthalmology.
8.30 p.m.—B.M.A. Hall: Professor F. Wood Jones, Lecture II.

Wednesday, November 15, 1933.

9.30 to 11 a.m.—Women's Hospital: Dr. B. Milne Sutherland, "Puerperal Sepsis".
11.15 a.m. to 12.45 p.m.—Women's Hospital: Dr. W. Ivon Hayes, "Ante-Partum Hæmorrhages".
2.30 to 5 p.m.—Melbourne Hospital: Demonstration of the newer anæsthetics, arranged by Section of Anæsthesia.
2.30 p.m.—Dr. F. W. Green: "Pre-Medication".
3 p.m.—Dr. G. L. Lilies: (*a*) "Use of Carbon Dioxide"; (*b*) "Spinal and Caudal Anæsthesia".
3.30 p.m.—Dr. H. C. Disher: "Gas Anæsthesia".
4 p.m.—Dr. Geoffrey Kaye: "Endo-Tracheal Anæsthesia".
8.30 p.m.—B.M.A. Hall: Professor F. Wood Jones, Lecture III.

Thursday, November 16, 1933.

9.30 to 11 a.m.—Alfred Hospital: Dr. M. D. Silberberg, "Stages of Mitral Stenosis".
11.15 a.m. to 12.45 p.m.—Alfred Hospital: Dr. Hugh Trumble, "The Rôle of Surgery in the Treatment of Pulmonary Tuberculosis".
2.30 to 5 p.m.—Infectious Diseases Hospital, Fairfield: Dr. F. V. Scholes and staff, demonstration of infectious diseases cases.

Friday, November 17, 1933.

9.30 to 11 a.m.—Children's Hospital: Dr. J. W. Grieve, "Diagnostic Difficulties".
11.15 a.m. to 12.45 p.m.—Children's Hospital: Dr. D. Officer Brown, "Treatment of Some Common Surgical Conditions of the Lower Limbs".
2.15 to 3.30 p.m.—Melbourne Hospital: Dr. S. V. Sewell, "Increased Intracranial Tension".
3.30 to 5 p.m.—Melbourne Hospital: Dr. W. Allan Hailes, selected cases from septic wards.

Saturday, November 18, 1933.

9.30 to 11 a.m.—Alfred Hospital: Dr. R. S. Ellery, "Senile and Pre-Senile Mental States".

Monday, November 20, 1933.

9.30 to 11 a.m.—Children's Hospital: Dr. A. P. Derham, "Nutritional Problems of Infancy".
11.15 a.m. to 12.45 p.m.—Children's Hospital: Dr. W. G. D. Upjohn, "Intestinal Obstruction in Childhood".
2.15 to 3.30 p.m.—Melbourne Hospital: Dr. Konrad Hiller, clinical demonstration of medical cases.
3.30 to 5 p.m.—Melbourne Hospital: Dr. A. E. Coates, "Treatment of Chronic Ulcer of the Leg and Allied Conditions".
8.30 p.m.—B.M.A. Hall: Dr. A. C. D. Rivett, Lecture I.

Tuesday, November 21, 1933.

9.30 to 11 a.m.—Alfred Hospital: Dr. G. A. Birnie, "Gastric Hæmorrhage".
11.15 a.m. to 12.45 p.m.—Alfred Hospital: Dr. John Kennedy, "Surgery of Anus and Rectum in General Practice".

Wednesday, November 22, 1933.

9.30 to 11 a.m.—Alfred Hospital: Dr. Robert Fowler, "Clinical Significance of Female Sex Hormones".
11.15 a.m. to 12.45 p.m.—Alfred Hospital: Dr. J. M. Buchanan, "The Management of Disorders of the Menopause".
2.15 to 3.30 p.m.—Alfred Hospital: Dr. Ewen Downie, "Treatment of Complications of Diabetes—Gangrene, Retinitis, Neuritis *et cetera*".
3.30 to 5 p.m.—Alfred Hospital: Dr. Charles Sutherland, "Investigation and Treatment of Asthma".
8.30 p.m.—Children's Hospital: B.M.A. clinical meeting.

Thursday, November 23, 1933.

9.30 to 11 a.m.—Saint Vincent's Hospital: Dr. J. G. Hayden, "Treatment of the Anæmias".
11.15 a.m. to 12.45 p.m.—Saint Vincent's Hospital: Dr. J. Newman Morris, "Inflammatory Conditions of Bladder and Kidney".
2.15 to 3.30 p.m.—Melbourne Hospital: Dr. F. Blois Lawton, "Some Bowel Disorders".
3.30 to 5 p.m.—Melbourne Hospital: Dr. Victor Hurley, "The Diagnosis and Treatment of Hydatid Disease of Liver and Lung".

Friday, November 24, 1933.

9.30 to 11 a.m.—Melbourne Hospital: Dr. R. R. Wettenhall, demonstration of cases of skin disease, with remarks on diagnosis and treatment.
11.15 a.m. to 12.45 p.m.—Melbourne Hospital: Dr. H. F. Praagst, "Radiological Diagnosis of Diseases of Bone".
2.15 to 3.30 p.m.—Melbourne Hospital: Dr. Douglas Thomas, "Chronic Non-Tubercular Respiratory Infections".
3.30 to 5 p.m.—Melbourne Hospital: Dr. Euan Littlejohn, demonstration in fracture ward.
8.30 p.m.—B.M.A. Hall: Dr. A. C. D. Rivett, Lecture II.

The three lectures by Professor F. Wood Jones will be on "Errors of Development of Interest in Medical and Surgical Practice". A fee of one guinea will be charged for these lectures.

The lectures by Dr. A. C. D. Rivett will be on "Some Aspects of Natural Applied Science in Australia". No fee will be charged for these lectures.

POST-GRADUATE COURSE IN OBSTETRICS.

A POST-GRADUATE course in obstetrics will also be held from November 13 to 25, 1933, at the Women's Hospital, Melbourne.

The course will comprise attendance in all departments of the hospital, with special lectures and demonstrations by members of the staff. Arrangements have been made whereby a limited number of graduates will be able to

enter into residence at the hospital. The accommodation will be allotted in order of application. The fee for the course will be three guineas. Additional fee for accommodation at the hospital will be three guineas per week.

It is requested that those proposing to attend these courses and those wishing to take advantage of hospital accommodation should send their names and fee as soon as possible to the Honorary Secretary, 61, Collins Street, Melbourne.

The Royal Australasian College of Surgeons.

A MEETING of the Council of the Royal Australasian College of Surgeons was held on August 26, 1933. Sir Henry Newland, the President, occupied the chair.

The President referred to the loss which the College had sustained by the death of Robert Hamilton Russell, a founder of the College and its first Censor-in-Chief. It was decided that his name, together with that of the late George Adlington Syme and the late Robert Gordon Craig, should be perpetuated in the new College building.

H. B. Devine was elected to the position of Vice-President of the College to fill the vacancy in that position occasioned by the resignation of Sir Alexander MacCormick.

The following appointments were made by the Council:

Censor-in-Chief: Alan Newton.

Honorary Secretary: Julian Smith, Junior.

Honorary Assistant Secretary: G. R. A. Syme.

The vacancy in the number of elected members of the Council was filled by the appointment of Balcombe Quick for the remainder of the late R. Hamilton Russell's term of office.

New Fellows.

The following candidates were admitted to fellowship:

New South Wales.—Hugh Hunter Jamieson.

Queensland.—Bruce Toomba Mayes, Clive Lansdell Paine.

Victoria.—William Balfour Wishart.

The following junior members were admitted to fellowship:

New South Wales.—Clifford Stirling Colvin, Ernest John Egan, Joseph Steigrad, Philip Neville Walker-Taylor.

Queensland.—Patrick Leo O'Halloran.

South Australia.—Ian Ayliffe Hamilton.

Western Australia: Stanley Earles Craig.

College Building and Annual Meeting.

The official opening of the College building will take place in March, 1935.

The next annual general meeting of the College will be held in Adelaide on March 5, 6, 7 and 8, 1934.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on September 4, 1933.

The degree of Bachelor of Surgery (B.S.) was conferred *in absentia* on Carl Ernest Mitchelmore Gunther, M.B.

Professor W. J. Dakin was appointed as representative of the University on the Fauna Advisory Committee.

The Vice-Chancellor (Dr. R. S. Wallace) notified the Senate that he had accepted appointment as a member of the Board of Directors of the Royal Prince Alfred Hospital.

Gifts for the Department of Surgery were accepted with grateful thanks from Colonel F. A. Maguire of an engraving entitled "Council of the Royal College of Surgeons of England, 1927", and from Dr. F. P. Sandes of a selection of surgical books and journals.

The following appointments were approved:

Dr. G. E. Phillips, M.Sc., as Liston Wilson Fellow for a third year.

Mr. R. Walker as Cancer Research Technician at Saint Vincent's Hospital.

Correspondence.

CANCER.

SIR: In your able review of the cancer problem in your issue of August 12, 1933, the salient point to me is the query of the Australian pathologist *re* normal growth, tissue regeneration and healing. The natural answer to such a question is that normal tissue regeneration is under control while tumour development is tissue development without control. What is the nature of that normal control?

The unicellular organism is self-propagating but has no capacity for cell differentiation or cell coordination. Food and environment control its reproduction. In the multicellular, the non-fertilized ovum has no capacity for either self-propagation or differentiation. Any such capacity must come from some source external to the ovum. The spermatozoon supplies the stimulus which initiates cell multiplication, differentiation and coordination. This stimulus and control is necessary from the beginning to the end of life.

In the invertebrates the lymphatic is the only circulation and it reaches every cell. Its glands are the only possible source of energy for the tissues. It maintains the cell control which the spermatozoon initiated. The lymphatic action is necessarily specific. The lymphatic system represents the spermatozoon and the tissues the original cell—the ovum.

Thus between both the spermatozoon and the lymphatic glands, and the ovum and the tissues, there is present a reciprocal antagonism in which the products of the one are the stimuli to the other.

In the vertebrates provision has to be made for a more complex organism and the bloodstream with its glands and hormones replaces the direct lymphatic stimulus for the tissues. But every endocrine, digestive, eliminative or reproductive gland passes into the lymph system an internal secretion. By this arrangement the lymphatic system is enabled the more easily to control the increased tissue complexity. The reciprocal antagonism is now between the lymphatic glands and the other glands.

The endocrine glands have an external hormone secretion and an internal, which is passed into the lymphatic system, is there specifically reversed and returned to the blood as chalone to the hormone of the gland from which it originated. Thus the autocoids are produced with their hormones for each tissue cell and their chalones for each hormone.

Any loss of balance between hormone and chalone is reflected in the cell which they control. Lowered hormone content leads to lowered cell potency and also to a lowered chalone content. As the chalone is the antibody to the hormone, the less hormone the less chalone, and there is no loss of autocoid balance. But lowered chalone content of the autocoid with a normal hormone secretion means a loss of autocoid balance and so much uncontrolled hormone action.

The more the chalone loses its control, the greater the uncontrolled hormone cell stimulus and the greater the cell proliferation. Finally all control of cell regeneration is lost and the unrestrained hormone action produces a rapid cell proliferation with a steady return to its original embryonic form.

In the invertebrates there is no cancer, for although the lymphatic glands may fail, there is present no hormone to produce cell proliferation. Cancer readily extends through the lymphatic system which has lost its specificity for the cell involved. Naturally the proliferation is strictly confined to the cell type in which it originated. The more vigorous the patient, the more active the hormone and the more virulent the neoplasm.

Cancer, then, may be defined as a gland imbalance produced by the loss of the normal control of cell regeneration and the unrestrained proliferation induced by uncontrolled hormone action. The balance is seen in a healing wound in which the regeneration is absolutely controlled.

Yours, etc.,
D. MONTGOMERIE PATON.

Malvern,
Victoria,
August 22, 1933.

SHALL THE TONSIL BE OUR TOTEM?

SIR: By coincidence, on the same morning as I opened my journal containing Dr. Ellery's letter of August 6, I received the following, which I think I may be excused for repeating.

From Regius Professor of Physic,
University of Cambridge,
July 26, 1933.

Dear Dr. Pern:

Many thanks for your letter and reprint. It is interesting to find that in 1924 you were already on the track of metabolic questions, which in those days were being overlooked. I am sure the question of infections of low grade virulence are of special importance in the production of thyroid disorders and the recent work on the output of calcium in hyperthyroidism fits in with your general conceptions.

Yours truly,
W. Langdon Brown.

Dr. Ellery's letter is an excellent piece of satire and I have no doubt that he derived a great deal of amusement and pride in producing it, but he cannot yet have arrived at the age of discretion or he would have learnt that such indulgences are apt to play the part of a boomerang in the hands of a novice and do more harm to himself than the individual he is aiming at. Unfortunately for Dr. Ellery, it can never be erased, but must stand for all time as a hall-mark of his mind. Criticisms are of great value and nobody appreciates them more than those who are trying to get at the root of our medical problems, but letters like Dr. Ellery's are wasting good space, yet at the same time they may be of some use in helping to focus attention on a subject that it is still going to take some years to be universally accepted. Time proves the truth or otherwise of most things. There is nothing so far that I have written in the past that has proved fallacious and the future has more yet to reveal. Truth is such a solid rock and has such firm foundations that it can withstand all the kicks and gibes that can be expended on it. I hope the next time I see Dr. Ellery in print, it will be to produce something more worthy of his pen.

Yours, etc.,
SYDNEY PERN.

Melbourne,
August 28, 1933.

A PROTEST.

SIR: To misquote Juvenal: *si natura negat, facit indignatio verba.* In a recent lecture that was published in this journal, Dr. Cilento stated that, with the exception of a small moiety of south Victoria, the whole of our country lies nearer to the equator than does any part of Europe. Now Dr. Cilento is a Commonwealth official, and therefore no Tasmanian would expect him to be aware that this State is a part of Australia. Years ago Commonwealth governments did their full duty by us when they conferred on us such inestimable benefits as the tariff, the arbitration court and the *Navigation Act*. Since then their successors and their officials have forgotten all about us, except when elections are approaching or when our unreasonable squirmings under the pressure of our blessings disturb their well-earned repose.

Nevertheless, Dr. Cilento's lecture will be eagerly read by school children the world over, and these children will have been taught that Australia includes Tasmania. His statement will therefore inevitably cause them to doubt their teachers' accuracy or omniscience. The possible results of such doubt are too dreadful to contemplate.

With less excuse, Dr. Elinor S. Hunt, of Sydney, and, alas, my old friend Dr. Leslie J. J. Nye, of Brisbane (*et tu, Brute!*), have tended to confirm the erroneous impression given by Dr. Cilento. Both these doctors have recently published in this journal articles containing medical statistics from all the Australian States except Tasmania.

I am unwilling to believe that our mainland colleagues find it necessary to dissemble their love for us, but I submit that when they thus ignore our existence they come perilously close to kicking us downstairs.

But perhaps I do them wrong. Can it be that they are reluctant to recognize as a component part of Australia a State whose governments have for years past been insufficiently enlightened to appoint a medical man as chief health officer?

Yours, etc.,
ERIC JEFFREY.

Lower Sandy Bay,
Tasmania,
August 31, 1933.

THE TREATMENT OF PNEUMONIA.

SIR: The remarks on oxygen administration by Dr. Gordon Hislop in his otherwise excellent article on the treatment of pneumonia (THE MEDICAL JOURNAL OF AUSTRALIA, September 2, 1933, page 322) call for some comment. The portable oxygen tent which he mentions is not satisfactory. Experiments in my department have shown that it requires at least ten litres of oxygen per minute to maintain an effective concentration (40%) of oxygen by this method. At this rate the largest size of cylinder (100 cubic feet) would last only about four and a half hours—barely time for the priest and family lawyer to perform their offices. Since oxygen administration must be continuous for so long as the pulmonary lesion prevents normal oxygenation of the arterial blood, it can readily be seen that the cost and transport difficulties would be considerable if this method is to be used effectively.

The second point is with regard to the nasal catheter. Seeing that with this method half the oxygen (during the expiration phase) is wasted, it is doubtful whether two litres per minute would be an effective rate in a severe case in an adult. Yet even this rate causes almost intolerable discomfort. From personal experience I can say that one's naso-pharynx feels like the interior of a hive of angry bees. Moreover, experiments have shown that the nasal catheter is no more efficient than the old-fashioned funnel, which, though wasteful, is effective if a sufficiently fast rate of oxygen flow (three to four litres per minute) is used.

Warming oxygen is, *per se*, of no consequence. The important point is that it should be saturated with water vapour at or about body temperature. Failing this, it may cause uncomfortable desiccation of the mucous membranes if administered in effective quantities.

It is surprising that Dr. Hislop makes no mention of the Haldane oxygen administration apparatus. This is economical because it conserves oxygen during the expiratory phase and, for rates of oxygen flow up to about three litres per minute, highly efficient. Meakins has shown that, with this apparatus, two litres of oxygen per minute are sufficient to maintain normal arterial oxygen saturation in nearly all cases of pneumonia. Meakins and I have used this apparatus in a large number of cases and have never known patients to object to the mask, provided the oxygen be turned on before the mask is applied.

Modern oxygen tents are now being extensively used in America. They differ from the local article in being made of rubber-proofed fabric and in having an external purifying circuit. They are efficient, reasonably economical, but somewhat cumbersome to transport.

The apparatus devised by Gilchrist and myself (*Edinburgh Medical Journal*, May, 1925) is simple, efficient and easily transportable. It is now being made locally by the Commonwealth Oxygen Company. It affords maximal efficiency with the minimum of discomfort to the patient.

Many other points might be discussed at length, but scarcely fall within the scope of the present letter. One would therefore conclude with an aphorism—the outstanding clinical criterion of effective oxygen administration is a substantial fall in pulse rate. Failure of this to occur may be due to: (i) inefficient administration, (ii) oxygen lack of the "stagnant" or the "anæmic" type, (iii) asphyxial heart-block.

Yours, etc.,

H. WHITRIDGE DAVIES.

Department of Physiology,
University of Sydney,
Undated.

Obituary.

ALBERT CURTIS.

WE regret to announce the death of Dr. Albert Curtis, which occurred on September 13, 1933, at Melbourne, Victoria.

LINDSAY WALKER CRAIG.

WE regret to announce the death of Dr. Lindsay Walker Craig, which occurred on September 15, 1933, at West Brunswick, Victoria.

Diary for the Month.

SEPT. 26.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 27.—Victorian Branch, B.M.A.: Council.
SEPT. 28.—South Australian Branch, B.M.A.: Branch.
SEPT. 28.—New South Wales Branch, B.M.A.: Branch.
OCT. 3.—New South Wales Branch, B.M.A.: Council.
OCT. 4.—Western Australian Branch, B.M.A.: Council.
OCT. 4.—Victorian Branch, B.M.A.: Branch.
OCT. 5.—South Australian Branch, B.M.A.: Council.
OCT. 6.—Queensland Branch, B.M.A.: Branch.
OCT. 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
OCT. 13.—Queensland Branch, B.M.A.: Council.
OCT. 17.—New South Wales Branch, B.M.A.: Ethics Committee.
OCT. 18.—Western Australian Branch, B.M.A.: Branch.
OCT. 18.—Victorian Branch, B.M.A.: Clinical Meeting.
OCT. 19.—New South Wales Branch, B.M.A.: Clinical Meeting.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought etc., see "Advertiser", pages xvi, xvii and xix.

AUSTIN HOSPITAL FOR CHRONIC DISEASES, HEIDELBERG, VICTORIA: Honorary Officers.

CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.

DIRECTOR-GENERAL OF HEALTH, CANBERRA, F.C.T.: Temporary Relieving Medical Officer.

GYMPIE GENERAL HOSPITAL, GYMPIE, QUEENSLAND: Junior Resident Medical Officer.

HOBART PUBLIC HOSPITAL, HOBART, TASMANIA: Junior Resident Medical Officers.

LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officers.

PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Resident Medical Officers.

ROYAL PRINCE ALFRED HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Officers.

THE ROYAL SOCIETY FOR THE WELFARE OF MOTHERS AND BABIES, SYDNEY, NEW SOUTH WALES: Junior Honorary Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
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